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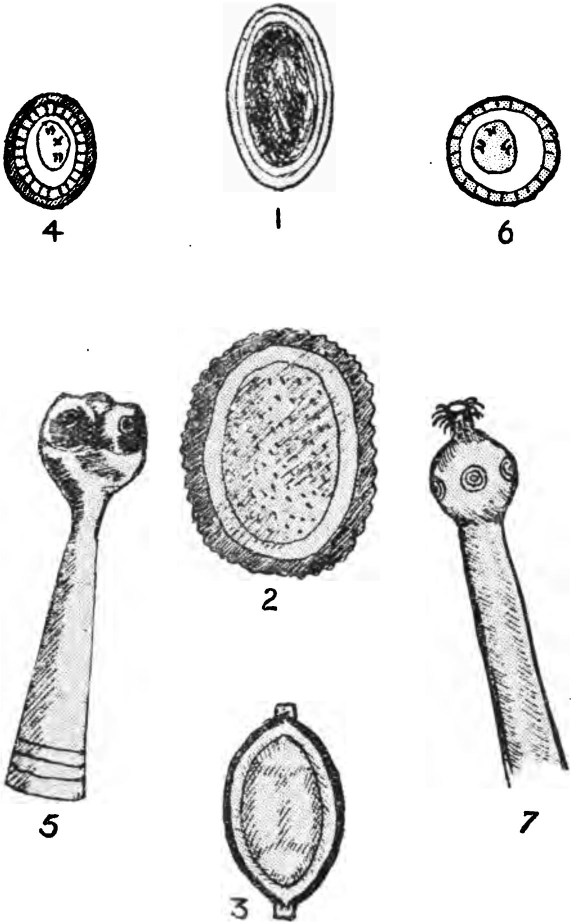

Cestodes and Nematodes.

1, Ovum of *Oxyuris vermicularis;* 2, Ovum of *Ascaris lumbricoides;* 3, Ovum of *Trichocephalus dispar;* 4, Ovum of *Tænia saginata;* 5, Scolex of *T. saginata;* 6, Ovum of *Tænia solium;* 7, Scolex of *T. solium.*

AIDS

TO

TROPICAL MEDICINE

BY

GILBERT E. BROOKE

M.A. CANTAB., L.R.C.P. EDIN., D.P.H., F.R.G.S.

PORT HEALTH OFFICER, SINGAPORE
LECTURER IN HYGIENE AND EXAMINER IN CHEMISTRY AND PHYSICS
TO THE MEDICAL SCHOOL OF THE STRAITS SETTLEMENTS
FORMERLY GOVERNMENT MEDICAL OFFICER AND DISTRICT COMMISSIONER,
TURKS AND CAICOS ISLANDS, WEST INDIES

AUTHOR OF
'A HYGIENE READER FOR SCHOOLS,' 'TROPICAL MEDICINE,
HYGIENE, AND PARASITOLOGY,' ETC.

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PREFACE

THE number of openings for medical men in the tropical portions of our Empire, the admission of cases of tropical disease by the agency of our increasing commerce, and the recognition of grave colonial and international sanitary questions, have all served to give an impetus to the study of tropical diseases at home.

The concrete results of this impetus have been manifested by the establishment of Schools of Tropical Medicine both in London and at Liverpool, as well as by the institution of Diplomas in Tropical Medicine to be conferred after due examination by several examining Boards.

The present small volume does not in any way aim at providing a text-book on the subject, but is rather intended to form a student's note-book, in which a concise and up-to-date summary of the chief points may be found. To attain this object, everything not deemed essential has been omitted, and theoretical considerations have been excluded as much as possible.

For convenience of reference the arrangement of subject-matter is strictly alphabetical throughout.

Some of the results of the Royal Society Commission on *Malta fever* have been incorporated.

It is hoped that the inclusion of sections on *Seven* *day fever*, *Epidemic dropsy*, and *Infectious jaundice* (as described by Rogers and by Sandwith), will add to the value of the book.

The Milroy Lectures on *Kala-azar* by Rogers, and the recent exhaustive treatise on *Trypanosomiasis* by Laveran and Mesnil, have been consulted in order to bring well up to date the sections which deal with those two diseases respectively.

GILBERT E. BROOKE.

BATH,
May, 1908.

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AIDS TO

TROPICAL MEDICINE

I

AINHUM

Definition.—A progressive constriction of one or more toes—usually the little toes.

Synonym.—Ring-toe.

Distribution.—It occurs chiefly amongst the negroes of West Africa and of the West Indies.

It has also been reported from India, Syria, Brazil, North Africa, British Guiana, Madagascar, and the Pacific Islands.

History.—*Clarke* was the first to call attention, in 1860, to this disease, as found on the West Coast.

Etiology.—The exciting cause is not known.

Heredity is apparently a predisposing cause; and it is possible, also, that the irritation of sharp grasses on the naked feet may have something to do with it.

Symptoms.—There is an early thickening of the digito-plantar fold of one little toe, and occasionally of both.

In rare instances it is noted on other toes.

The thickening becomes constricted until a groove completely surrounds the toe, eventually leading to automatic amputation.

Ulceration is not constant. Unless ulceration is present, pain is rare.

Treatment.—If there is any family history, the skin should be hardened and local irritation avoided.

It has been recommended as advisable to sever the constricting fibrous band at an early stage, but the proceeding is of doubtful value.

Amputation will eventually be necessary.

Pathology.—There is an early and slight œdema.

The subcutaneous adipose tissue is largely increased. The bone is partly absorbed. The constricting ring is formed of fibrous tissue, and as this increases tactile sense is gradually lost.

II

ANKYLOSTOMIASIS

Definition.—An anæmic cachexia induced by an intestinal nematode—the *Ankylostoma duodenale* (Dubini, 1843).

Synonyms.—

1. *Of the Parasite*—
   Strongylus duodenalis; Dochmius duodenalis.
2. *Of the Disease*—
   Egyptian chlorosis; Ceylon anæmia; Miner's anæmia.

Geographical Distribution.—It was first reported from Brazil by *Piso* in 1864.

It appeared in epidemic form amongst the staff working on the St. Gothard Tunnel.

Its distribution is world-wide.

It is endemic chiefly in the tropics. The following are the chief endemic centres: Egypt, North Africa, Abyssinia, West and South Africa, Madagascar, Mauritius, India, Assam, Ceylon, Malaya, Australia, New Guinea, Fiji, Philippines, Japan, Southern States, Central and most of South America.

Etiology.—The parasite belongs to—

Order—Nematoda.
Family—Strongylidæ.
Genus—Ankylostoma.

Its *Morphology* is—

Body cylindrical and pinkish.

Mouth $\begin{cases} \text{4 hooked teeth ventral.} \\ \text{2 ,, ,, dorsal.} \end{cases}$

Males: 6 to 8 millimetres long by 0·5 millimetre broad. At the posterior end is a bell-shaped bursa, in which is a bottle-shaped penis and two long spicules.

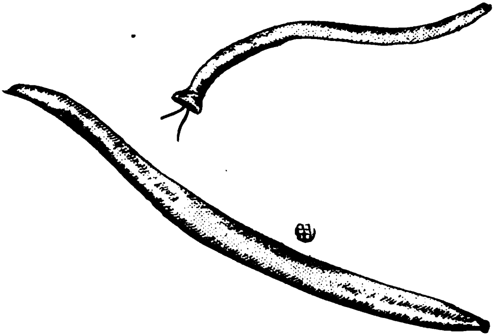

FIG. 1.—*A. DUODENALE.*

Male and Female in position of Copulation.

Females: 10 to 12 millimetres long by 1 millimetre broad. Small spine at posterior end. Vulva at junction of middle and hinder third.

In copulation resembles Greek γ.

Ova elliptical, thin shells, segmented yolk; 60 μ long by 40 μ broad.

Habitat.—Lives in duodenum, more rarely in jejunum. Fastens on to mucous membrane with its teeth. Thousands may be present. F. : M. :: 3 : 1.

Development.—Ova develop in fæcal matter, but die in fresh water. In one or two days rhabditiform larvæ (0·21 millimetre long) are liberated from the shell, and continue to grow to 0·6 millimetre, moulting twice. At this stage they will remain alive in water for three or four months.

Infection.—All races, sexes, and ages are liable. Infection occurs—

1. *By the Mouth.*—When ingested they soon leave the stomach. They moult on the seventh and fifteenth day, when an oval capsule is formed. They are mature in four or five weeks and copulate.

2. *By the Skin.*—The larvæ, in contaminated soil, enter the skin of coolies and other naked natives by means of a follicle. This often causes a local dermatitis, known as 'Coolie Itch.' It then journeys, via veins, heart, arteries, air-vesicles, bronchi, trachea, œsophagus, and stomach, to the duodenum.

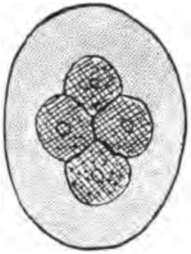

FIG. 2.—OVUM OF *A. DUODENALE.*

Symptoms.—Many of these nematodes may be present without symptoms. Clinically the condition is recognized by—

1. Ova in fæces.
2. Anæmia.
3. Dyspepsia.

The anæmia may be extreme, and death occur from exhaustion, diarrhœa, or intercurrent disease.

If treated, the prognosis is favourable up to the cure of 90 per cent.

Treatment.—Thymol is the best drug. Give liquid diet for two days.

Then a purge.

Then thymol, gr. xx., on an empty stomach every hour for three doses.

Care should be taken when using thymol:

1. Lie down for some hours afterwards.
2. Avoid alcohol, glycerine, etc., and other solvents of the drug, for some time after taking.
3. Contra-indications are—
   - (*a*) Advanced cases.
   - (*b*) Gastritis.
   - (*c*) Dysentery.
   - (*d*) Nephritis.

Pathology.—There is emaciation and anæmia of most organs.

The lungs are œdematous.

The stomach shows chronic catarrh.

The jejunum shows petechiæ.

The parenchyma cells of liver and kidneys sometimes contain hæmatoidin.

III

BERI-BERI

Definition.—A specific multiple neuritis characterized by œdema, paresis, loss of knee-jerks, and frequent implication of the phrenic and pneumogastric nerves.

Geographical Distribution.—It has a wide tropical and subtropical distribution, with sporadic incidence in temperate regions.

The three chief endemic centres are :

(*a*) Malaya.
(*b*) Japan (main island).
(*c*) Brazil (coast and interior).

Other centres are : Burma, Siam, South China and Hong-Kong, the Philippines, East and West Coasts of Africa, Madagascar, Mauritius, Central America, Cuba, and some other West Indian Islands.

Etiology.—The specific agent has yet to be found. The theories have been multitudinous. They may chiefly be summed up under the following heads :

1. Infected fish.
2. Infected rice.
3. Nitrogen starvation.
4. Arsenical poisoning.
5. Oxalate poisoning.
6. A blood protozoon.
7. A bacillary origin.
8. A volatile toxin.

	Beri-beri.	Alcoholic Neuritis.	Locomotor Ataxy.
Sex	Usually males	Often females	Usually males
Age	Eighteen to thirty	Middle age	Thirty to forty
History	Nothing, or	Elbow bending	Syphilis
	Previous attack	Muscular tenderness	Lightning pains Argyll-Robertson pupils
Tenderness	Of calf muscles extreme	General	Nil
Sensation	Impaired or lost	Absent or hypersensitive	Plantar anæsthesia
Trophic	Muscular wasting	Muscular wasting	± Perforating ulcer of foot
Œdema	Always pre-tibial Sometimes general	Probable	± Of feet
Knee-jerks	Lost	Lost	Lost
Ankle-clonus	—	—	Nil
Urine	Chlorides (−) Oxalates (++)	Albumin	± Incontinence
Gait	Foot raised high Ankle drops	Toes raised Foot slaps flat, like a flail	Leg thrown violently Foot raised high
Course	Toe first on ground Acute, chronic, or relapsing	Recovery slow	Heel first to ground Matter of years Recovery improbable
Pathology	Peripheral nerves affected Sheath and axis cylinder disappear	Extensors mostly involved	Degeneration of posterior roots and dorsal columns of cord

Treatment.—No drug with specific action has yet been found.

The treatment must be largely routine and symptomatic.

The patient should be removed to cool and airy quarters.

The diet should be digestible and nutritious.

The bowels should be kept open.

For the paresis, strychnine, gr. $\frac{1}{50}$, should be injected into each thigh daily.

For the œdema, a diuretic, such as—

℞.	Liq. ammon. acet.... ...	ʒi
	Pot. nit.	gr. x.
	Pot. acet.	gr. xv.
	Aq. camph.	ad ℥i.
	M. Ft. mist. Sig.: ℥i. t. d. s.	

Heart failure will demand digitalis and stimulants.

When the hyperæsthesia has departed, faradization and massage will be of use in treating the muscular atrophy.

A sea-voyage and tonics will be indicated during convalescence.

Pathology.—Rigor mortis is short, and a p.m. rise of temperature is sometimes seen.

The heart will be enlarged and the right ventricle dilated.

The lungs are œdematous.

The peripheral nerves show inflammatory degeneration. The sheath and axis cylinders break up and disappear.

The nerve trunks do not suffer as a rule, but branches to muscles and arteries, the splanchnics, and the solar and renal plexuses, all participate in the degeneration.

The vagus and its branches are involved in acute cases.

Its *Morphology* is—

Males : 12 to 14 millimetres long.
Lateral edges curve ventrally, forming the gynæcophoric canal.
Oral and ventral suckers at anterior end, and close together.
On dorsum, posteriorly, are spinous papillæ to grasp vein walls.
Excretory pore dorsal and posterior.
Genital pore ventral and anterior, behind ventral sucker. There is no penis.

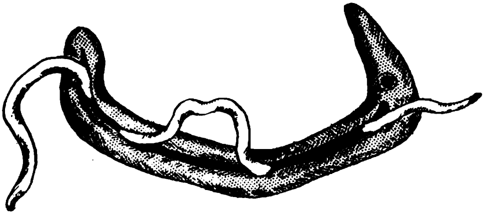

FIG. 3.—*S. HÆMATOBIUM.*
Female in Gynæcophoric Canal of Male.

Females : Filiform. Length, 20 millimetres.
Suckers and pores as in male.
For coitus, the female lies in the gynæcophoric canal of the male, the genital pores being merely approximated.

Ova : These are oval, yellowish, and slightly transparent, enclosed in a thin shell with a terminal spine (sometimes lateral).
They are 0·16 millimetre long by 0·06 millimetre broad, and contain a ciliated embryo (miracidium).

The *Habitat* of the adult is in the *portal vein*, with its tributary branches, especially those of the bladder and rectum. The adults pair at puberty, and descend to the bladder and rectum to deposit their eggs.

As many as 300 adults have been found in one patient.

The small veins and capillaries are torn and bored through by the ova, which thus gain the urine or fæces.

The *Development* of the embryo takes place only in water. On reaching this, either with the urine or fæces, the eggshell is ruptured and the miracidium swims away. There is possibly a small crustacean to act as intermediate host for further development, just as guinea-worm embryos develop in *Cyclops*, but this is not yet worked out.

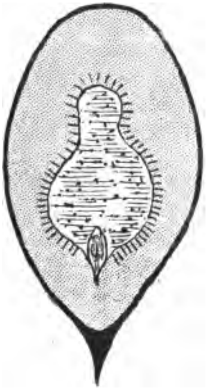

FIG. 4.—OVUM OF *S. HÆMATOBIUM.*

Human *Infection* very likely may take place by two channels, as in the case of ankylostomiasis :

1. By the mouth.
2. By the skin.

Native males, especially country-folk, are most subject to attack.

In Egypt the incidence is greatest when the Nile is up.

The *incubation period* is from four weeks to four months.

The adult worm may live for a number of years.

Symptoms.—In some cases these may be almost nil. Hæmaturia is the chief symptom. Micturition is frequent, with slight irritation, but very little pain.

The last few drops passed are blood-stained and contain ova.

The condition may last for months or years, and, in the event of no re-infection, may cease on the death of the adult parasites.

In some cases cystitis may be set up and the urine become alkaline, blood-stained, and purulent.

In 20 to 30 per cent. of the infections, urethral fistulæ and sinuses are found. Roof fistulæ present away from the middle line. Floor fistulæ are more painful, often cause stricture, and present in the middle line.

If the rectum is involved, troublesome excrescences will be found, and hæmorrhoidal or dysenteric conditions may be simulated.

Invasion of liver or lungs usually produces no symptoms.

Although often apparently trivial, the condition should be regarded with attention, since serious anæmia and emaciation frequently result, and death occurs in about 6 per cent. of cases either from exhaustion, pyæmia, uræmia, or intercurrent disease.

Treatment.—There is no means of directly dealing with the adults in the portal system.

Symptoms should be dealt with on ordinary surgical lines as they arise.

For the early hæmaturia the bladder should be occasionally washed out with boracic lotions.

Male fern (liquid extract) should be given in 15 minim doses three times daily, as a routine treatment. It often acts like a charm in controlling the hæmorrhage, and sometimes also appears to cut short the disease.

Pathology.—The *bladder* is the usual site for invasion. The walls are hypertrophied. The mucous membrane is reddened and swollen.

Excrescences containing eggs are plentiful, especially in the trigone.

There is an immense deposit of ova in the submucosa.

Calculi are frequent, consisting of oxalate of lime with a nucleus of ova.

The *vesiculæ seminales* and *prostate* are frequently invaded.

The *rectum* and *vagina* are less favourite sites, and will show similar morbid conditions to those in the bladder.

Ova have also been found in the following places: Left ventricle; mesenteric glands; lungs; liver; gallstones; and in the cutis.

V

BLACKWATER FEVER

Definition.—A tropical fever of unknown origin, characterized by the occurrence of free hæmoglobin in the urine.

Synonyms.—Hæmoglobinuric fever ; bilious remittent fever ; West African fever.

Geographical Distribution.—French medical officers on the West Coast of Africa mention the disease as early as 1820. It is most prevalent on the Gold Coast, in the Gaboon district, on the Congo, and in Central Africa.

Cases have occurred in East Africa, Madagascar, Mauritius, India, Assam, Cochin China, Java, New Guinea, Penang, Palestine, the Southern States of U.S.A., the West Indies, Central America, and many parts of South America. It has also been observed in many countries on the Mediterranean littoral.

Etiology.—The specific factor has not been discovered. Any race may be attacked, but long residence in an endemic area seems to convey immunity, in the case of natives especially.

There are three theories :

1. *That it is malarial.* This is the view taken by many, and supported by *Scheube*, *Plehn*, and others, on the ground that—

(*a*) It occurs largely in malarial centres.

(*b*) Malarial parasites are often found in the blood of these patients.

(*c*) That large mononuclear leucocytes are increased in numbers.

(*d*) That pigmented large mononuclears are commonly found in the blood.

Note.—These evidences of concurrent or propinquitous malaria are very commonly found also in the case of kala-azar and of trypanosomiasis, and are therefore not conclusive.

2. *That it is a quinine-poisoning.* This is supported by *Koch*, *Stephens*, *Christophers*, and others, on the ground that, in most cases, the symptoms develop after the administration of quinine.

Note.—This is not a sound argument. Of the numberless thousands who take quinine (often in large doses) throughout the world, would such a toxic idiosyncrasy be so limited? It is unlikely.

3. *That it has a specific origin.* The symptomatic analogies of this disease to the 'Texas fever' of cattle are so striking that it would not be unreasonable to suppose that blackwater fever may be due to a blood parasite, somewhat similar to the *Pyrosoma bigeminum*, which is answerable for that disease in cattle.

Note.—This seems the most probable view. In 1,000 cases of Texas fever hæmoglobinuria will be almost or entirely constant; in a similar number of malarial cases such a symptom will almost certainly be absent.

Symptoms.—Prodromata are rare. There is a rigor, with a rapid rise of temperature, generally accompanied by a headache, some muscular pains, and vomiting.

For two or three days the temperature is intermittent, with a constant tendency to rise.

The maximum is reached on the second or third day, and then only for the first time does the hæmoglobinuria appear.

The urine is scanty and of high specific gravity; contains free hæmoglobin, but no corpuscles, although renal and vesical epithelial cells are generally found.

A general jaundice occurs soon after the onset of the hæmoglobinuria.

The liver and spleen are considerably enlarged, with pain over both regions.

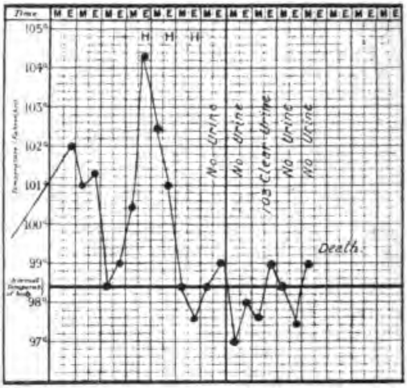

FIG. 5.—CHART OF BLACKWATER FEVER.
(H. signifies Hæmoglobinuria.)

After two or three days of these symptoms, the temperature falls, the symptoms abate, and convalescence will soon be established, but with obstinate albuminuria.

In fatal cases the temperature may rise again, and death occur from hyperpyrexia, coma, or convulsions; or, on the other hand, the temperature may remain normal or subnormal, anuria supervene, and death follow uræmic symptoms.

The blood is thin, and anæmia marked.

The case mortality varies from 10 to 50 per cent.

Diagnosis.—Yellow fever and infectious jaundice should be excluded.

Treatment.—Quinine is useless. Begin with a purge; apply counter-irritants to the epigastrium; give ice to suck; exhibit alcohol and nutrient enemata if necessary. With subnormal temperature and collapse apply hot bottles, and give warm saline intravenous injections.

Pathology.—A sudden and very large destruction of red corpuscles occurs. The pigment is eliminated mostly by the kidneys, some remaining in liver and skin.

The free hæmoglobin causes a disintegration of kidney epithelium, or even an extensive nephritis.

The corpuscles show—

(*a*) Oligocythemia;
(*b*) Poikilocytosis;
(*c*) Chlorocytosis.

The spleen is enlarged, soft, and dark.

The liver is enlarged and jaundiced.

VI

CHOLERA

Definition.—An acute specific disease characterized by vomiting, purging, cramps, anuria, and collapse.

Synonym.—Asiatic cholera.

Geographical Distribution.—The endemic habitat *par excellence* has been, since remote ages, India. From thence it has spread widely, by trade and pilgrim routes, to almost all parts of the world.

Nearly every region in the habitable globe has been visited at some time or another with the following exceptions: Australia, New Zealand, some of the Pacific Islands, the Azores, Ascension, St. Helena, the West Coast of Africa, the Cape, Bermuda, Chili, Peru, the Orkneys and Shetlands, the Faroe Islands, Iceland, and (strangely) the Andaman Islands.

During the nineteenth century there have been seven invasions of Europe.

Etiology.—The disease is an infection by *Spirillum choleræ* (Koch), discovered by Koch in Egypt in 1883.

A synonym is the 'comma bacillus.' The following are the characteristics of the organism:

They are curved like a German comma.

Length, 1·5 to 2 μ.

Thickness, 0·5 μ.

Motile.

Liquefy gelatin (fifty hours).

Flagellum, terminal and single.

No spores.

Decolorized by Gram.

Grow on ordinary media, except potato.

Killed in an hour by 55° C.

Drying kills generally in two or three minutes.

The disease is conveyed from person to person by contaminated food, drink, fingers, or utensils.

It is seldom dust-borne.

Flies will mechanically infect food with great rapidity if a focus of fæcal infection is of easy access.

The lower animals are all immune, unless intestinal peristalsis has been artificially inhibited before infection.

The profound prostration and collapse so inseparable from the disease are due to toxins elaborated by the vibrios. Like toxic phenomena can be induced in animals by injection of dead vibrios.

The following are two valuable *diagnostic reactions:*

1. *Cholera-Red Reaction.*—Depends on the fact that, if H_2SO_4 be added to indol+a nitrite, then a red-coloured nitroso-indol body results.

The *S. choleræ*, after twenty-four hours' culture in peptone broth at 37° C., forms both nitrites and indol, and will therefore turn pink on the addition of a drop or two of H_2SO_4.

A culture of Finkler and Prior's vibrio gives no similar reaction in twenty-four hours.

B. coli communis forms indol, but not nitrite.

2. *Starch-Acid Reaction.*—A slightly alkaline, peptone broth medium, to which starch and litmus have been added, should be used for a twenty-four hours' cultivation of the *S. choleræ* at 37° C. An acid reaction will occur within that time.

The Finkler-Prior vibrio gives a feeble acid reaction by the third day.

B. colic communis and *B. typhosus* give no acid reaction.

Symptoms.—The *incubation period* is usually from one to twenty-four hours.

The sudden onset begins with vomiting and a watery, bilious diarrhœa. These symptoms rapidly become aggravated. The retching and vomiting become frequent, and the stools become colourless, with white flocculi (rice-water stools).

Dreadful cramps of legs and abdomen are a painful and constant symptom.

The face is at first anxious, and soon becomes very pinched and sunken, giving a typical and pathognomonic facies.

The skin of fingers and toes becomes shrivelled and livid (washerwoman's fingers). The voice becomes husky, and finally a hollow whisper.

There is complete suppression of urine. The body is clammy and cold.

This is the algide or collapse stage, and terminates either in death or reaction.

In the latter case the symptoms abate, the vomiting and cramps cease, urine is secreted, the stools become bilious, and convalescence is soon established.

In addition to the above typical cases, other types are seen.

An *ambulatory variety*, in which the symptoms may be slight and almost unnoticed, and which is a dangerous factor in the spread of the disease.

Fulminating cases also occur, in which death from toxæmia occurs before the declaration of the usual symptoms.

Pregnant women contracting cholera almost always abort.

The *case mortality* is from 40 to 90 per cent., and rarely below 60 per cent.

Treatment. — Astringents — such as chlorodyne, lead, opium, chalk, etc.—are frequently prescribed, but drugs are not of much use in cholera.

Salol is perhaps the most useful.

Stimulants will be needed constantly, either by mouth or hypodermic injection.

Hot bottles should be frequently applied in the algide stage.

The body fluids being depleted to the utmost limit, the most urgent indication is to replace them as promptly as possible. Rectal or subcutaneous injections are absorbed too slowly. Large intravenous injections of warm saline solution should therefore be given, and continued till the pulse is restored, and should be repeated if necessary.

Prophylaxis.—1. *Eucalyptus-oil* has a marked effect. Doses of ♏x. should be given twice daily to those exposed to infection.

2. *Haffkine's Anti-Cholera Virus.*—Inoculations are performed with cultures exalted by passage through the peritoneum of guinea-pigs, and the results have been fairly satisfactory.

Pathology.—The condition is one of acute toxæmia, and not of septicæmia.

The 'comma bacillus' is only found in the intestinal tract.

The lungs are small and collapsed; the abdominal viscera are congested.

The peritoneal coat of the intestine is dry and injected.

Within an hour or two of the attack, the contents of the small intestine resemble Camembert cheese.

Very soon typical 'rice-water' stools are found in large and small intestines. The mucous membrane is dull red or acutely congested.

The bladder is nearly empty if death has occurred during the algide stage.

VII

DENGUE

Definition.—A specific tropical fever characterized by severe muscular pains and a definite skin eruption.

Synonyms.—Dandy fever; break-bone fever.

Geographical Distribution.—Three pandemics have occurred. In 1780-1783 it spread from India to Java, Egypt, Zanzibar, and America.

In 1824-1828 it visited India, Burma, North and South America, and the West Indies.

In 1870-1873 it spread from Zanzibar to Port Saïd, India, Burma, Siam, Java, Sumatra, Mauritius, and the United States.

It is now found in most tropical countries, occurring in limited epidemics.

Etiology.—The specific micro-organism is not yet known, and is probably ultramicroscopical in size.

The intermediate host is almost certainly a mosquito.

Stegomyia fasciata and *Culex fatigans* seem both to be incriminated.

The disease can also be produced by intravenous injection of infected blood.

The incubation period is from one to three days.

Symptoms.—The onset is usually remarkably sudden.

Severe muscular and arthritic pains occur without warning.

The temperature rises, and within an hour or two the primary rash makes its appearance. It is erythematous, usually confined to the face and extremities; is frequently transient, and at most only lasts a day.

Meanwhile the urgent symptoms increase, with severe pains and headache, while the fever rises until about the third day, when a crisis occurs, with fall of temperature and relief of all the chief symptoms.

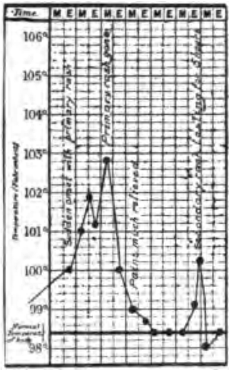

FIG. 6.—CHART OF DENGUE FEVER.

On the fifth to seventh day of the disease there is a slight secondary rise of fever, accompanied by a secondary eruption, which more resembles measles than does the scarlet fever type of the primary rash.

Pain returns at the same time.

Both fever and rash may be very transient, or may persist for a day or two.

If the rash has been at all marked, it will be followed by a slight furfuraceous desquamation.

Obstinate rheumatic pains often persist for some months.

Mental depression, sleeplessness, boils, etc., are occasional sequelæ.

The mortality is practically nil.

Treatment.—

℞. Aspirin.	gr. v.
Sp. chlorof.	♏x.
Tct. cardam. co.	♏xx.
Aq. chlorof.	ad ℥i.

M. Ft. mist. Sig.: ℥i. every 4 hours.

VIII

DHOBIE ITCH

Definition.—A common tropical body ringworm, generally confined to the neighbourhood of the crutch.

Distribution.—Common in the East.

Etiology.—It is a trichophyton infection, much like *Tinea circinata.* Microscopically the fungus much resembles the *Tinea ectothrix*, which is the cause of the latter disease.

Castellani considers it due to several different species.

Manson thinks *M. furfur* and *M. minutissimum* to be the causal agents. The disease is often spread by means of clothes.

Symptoms.—A raised red spot occurs, and spreads with an irregularly circular outline. Activity is chiefly confined to the spreading margin.

Various foci may coalesce until a large area is involved.

Desquamation occurs, but is very fine.

There is much itching, and the constant scratching generally leads to an eczematous condition which is frequently hard to cure.

Treatment.—In the early stages the following is a specific:

℞.	Chrysarobini	...	...	...	ʒi.
	Ol. caryophylli	...	...	...	♏x.
	Ung. paraffin.	...	...	...	℥i.

M. Ft. ung.

If the surface has become raw from scratching, then glycerine of boracic acid should be painted on three times daily, followed by a slight dusting with boracic powder, rest being enjoined.

IX

DIATHERMASIA

Definition.—An acute condition due to the effect of heat on the thermotaxic mechanism of a non-acclimatized or predisposed person, and characterized by hyperpyrexia and pulmonary congestion.

Synonyms.—Heat-stroke; insolation; heat-apoplexy; thermic fever; siriasis.

Geographical Distribution.—The condition most frequently occurs in the regions of the Red Sea, Persian Gulf, the provinces of Bengal and Lower Burma, Algeria, the valleys of the Nile, Mississippi, Amazon, and La Plata Rivers, and the hotter parts of the littoral of Queensland and the United States. It is unknown at a higher altitude than 600 feet, and never occurs in Europe.

Etiology.—A high external temperature causes hyperactivity of the heat-producing mechanism (*i.e.*, the trophic nerves) by paralyzing those parts of the thermotaxic centres which control the heat-discharge (*i.e.*, the vaso-motor nerves and sweat glands).

Predisposing causes will obviously be—non-acclimatization, intemperance, fatigue, and debilitating diseases.

Symptoms.—The attack may occur either by day or at night, and quickly becomes serious.

For a few hours lassitude, giddiness, headache, thirst, or frequent desire for micturition, may occur as prodromata.

The temperature mounts rapidly, sometimes even to 110° F. The face is cyanosed, and the conjunctivæ are injected.

There is coma or delirium, with embarrassed or stertorous breathing.

The skin is hot and dry, and the pulse thready, intermittent, and of low tension.

The pupils are variable and the reflexes sluggish.

Convulsions are common before death, which occurs (usually in a few hours) in about 75 per cent. of cases, and may be due to cerebral hæmorrhage, syncope, or respiratory failure.

The hyperpyrexia will serve to exclude other forms of coma when making a diagnosis.

Cerebro-spinal fever, and cerebral malaria should be excluded.

Treatment.—This should be as prompt as possible.

An immediate cold bath, with ice to the head, should be given.

Also a hypodermic of hyoscine, gr. $\frac{1}{75}$, to allay the cerebral hyperactivity.

When the pulse becomes thready, give also a hypodermic of digitalin, gr. $\frac{1}{100}$, and repeat in a quarter of an hour if necessary.

If the temperature falls to 103° F., wrap the patient in a blanket with hot bottles, with the hope that the grasp of the central nervous system on the thermotaxic mechanism is sufficiently restored to allow the sweat glands to act.

If it is not, and the temperature begins to rise again, then renew the cold-pack, etc., at once.

Avoid quinine and strychnine. Alcohol may be necessary when the temperature has fallen.

Be careful of relapses during convalescence. Light diet, open bowels, and bromides are the points to be noted. After a few weeks a change to a cooler climate should be insisted on.

Pathology.—The cerebral vessels and sinuses are intensely congested, and the ventricles usually contain serous fluid.

The heart shows rigid rigor mortis, and contains no clot.

The lungs are engorged with dark fluid blood.

The kidneys are congested, with the pyramids dark and prominent.

X

DISTOMIASIS

Definition.—An infection of man by various trematodes (other than the Bilharzia).

Trematodes of Man.—

1. *Gastrodiscus hominis.*
2. *Dicrocœlium lanceatum.*
3. *Cotylogonimus heterophyes.*
4. *Fasciola hepatica.*
5. *Fasciolopsis buski.*
6. *Opisthorchis noverca.*
7. *Opisthorchis sinensis.*
8. *Opisthorchis felineus.*
9. *Paragonimus westermani.*
10. *Schistosomum japonicum.*

The tables on pp. 36, 37 and 38 will show at a glance the chief characteristics of the above parasites, all of which are hermaphrodite except *S. japonicum*, in which, like the allied *S. hæmatobium* (Bilharzia), the sexes are distinct.

Symptoms.—These will, of course, depend on the habitat of the adult trematode in question.

Those which select the liver, bile-ducts and neighbourhood will frequently give rise to an interstitial hepatitis with subsequent hepatic atrophy. In this case jaundice and ascites will be present, and the ova probably be found in the stools. Those which dwell in the intestine may or may not cause anæmia and recurring diarrhœa.

The *P. westermani*, which lives in the lung, causes a chronic cough, urgent in the early morning. The expectoration is rusty brown (with occasional hæmoptysis), and contains many ova.

Treatment.—From their situation, it is obvious that but little can be done in the way of treatment, unless the parasite is located in the intestine, in which case some anthelmintics may be of use — such as thymol, male fern, pelleterine tannate, etc.

ADULT CHARACTERISTICS OF THE TREMATODES OF MAN.

	Size in Millimetres.	Normal Host.	Incidental Host.	Habitat.	Geographical Distribution.	Remarks.
Gastrodiscus hominis ...	6·5 × 3·5	Some Indian mammal	Man (twice)	Large intestine	India Assam	Small Reddish colour Opens at one end as an enlarged circular disc
Dicrocœlium lanceatum	9 × 2	Nearly all domestic animals	Man (seven times)	Bile-duct	Europe U.S.A. Siberia Algiers Egypt	Pointed at both ends, especially anteriorly
Cotylogonimus heterophyes	2 × 1	Dog	Cat Man (rare)	Small intestine	Europe Egypt	Smallest distoma of man
Fasciola hepatica ...	25 × 10	Sheep	Other herbivorous animals Man (twenty-three times)	Liver	Europe North Africa Afghanistan Thibet, etc.	Pathogenic to sheep Serious epidemics frequent

Fasciolopsis buski ...	50 × 10	Unknown	Man (eight times)	Intestine	China Sumatra Straits Settlements Assam India British Guiana	Largest distoma found in man
Opisthorchis noverca ...	11 × 2·5	Unknown	Man (occasionally)	Bile-ducts	India	Covered with spines
Opisthorchis sinensis ...	12 × 3	Dog Cat	Man (fairly common)	Bile-ducts and gall-bladder	India Mauritius China Japan	Transparent Contractile May occur in thousands Pathogenic
Opisthorchis felineus ...	10 × 1·5	Cat	Dog Fox Man (many times)	Gall-bladder and bile-ducts	Siberia	Pathogenic, but less mortality than *O. sinensis*
Paragonimus westermani (*Distoma ringeri*)	9 × 5	Dog	Cat Man Pig	Lung (rarely liver, testis, or brain)	China Japan Korea Mexico	Has scale-like spines Causes the endemic hæmoptysis of the East
Schistosomum japonicum	M. 10·5×0·5 F. 0·65×0·4	Cat	Man	Small mesenteric blood-vessels	China Japan	Pathogenic in man

CHARACTERISTICS OF TREMATODE OVA.

	Size in Millimetres.	Character.	Intermediate Host.
G. hominis	0·15 × 0·07	Oval	—
D. lanceatum	0·04 × 0·02	Oval Shell thick and yellow	? Slugs
C. heterophyes	0·03 × 0·02	Oval Thick shells	—
F. hepatica	0·13 × 0·07	Oval Shell brown and operculated	*Limnæus truncatulus*
F. buski	0·15 × 0·075	Oval Operculated	—
O. noverca	0·035 × 0·02	Oval Operculated	—
O. sinensis	0·03 × 0·015	Oval Shell thin, black, and operculated	—
O. felineus	0·03 × 0·01	Oval Operculated	? *Limnæus stagnalis*
P. westermani	0·09 × 0·05	Oval Shell thin and brown	—
S. japonicum	0·075 × 0·04	Oval and yellow No spine nor operculum	—

XI

DRACONTIASIS

Definition.—An endemic disease of the tropics, consisting of invasion by a nematode—the *Filaria medinensis* (Velsch, 1674).

Synonyms.—Guinea-worm; Medina-worm; *Dracunculus medinensis.*

Geographical Distribution.—The following are endemic centres:

The West Coast of Africa, Nubia, the Soudan, Red Sea littoral, Persian Gulf, Caspian Sea, Turkestan, West Coast of India from Bombay to Kutch, the Deccan, Rajputana, and Fiji.

Etiology.—The *morphology* of the *Filaria medinensis* is as follows:

The *males* are seldom seen, but have been found in infected monkeys.

The *females* are filiform, and attain a length of 50 to 80 centimetres or more. The cuticle is yellowish-white and firm. The mouth has two lips, behind which are six papillæ.

Posteriorly is a spine slanting ventrally, and 1 millimetre in length. The vulva and vagina are not known. Most of the body is taken up with the uterus.

The *habitat* of the adult female is in the connective tissue under the skin or between the muscles. Usually there is only one parasite, but as many as ten have been counted in one patient. They are capable of free progression, and never wound bloodvessels and nerves in their journeys. By a wonderful intuition they seek some part of the body which is most frequently in contact with water, and by this means the embryos can reach the element necessary for their further development.

The course of this *development* has been to some extent worked out. Contact with water causes the extrusion of several thousand embryos at a time, from the small skin ulcer made by the adult. These embryos become active, and live for some days in water. They are ingested by a minute crustacean—the *Cyclops quadricornis*—and grow somewhat. They become sluggish after three or four weeks, and are dead by the sixth week if nothing further happens. If the cyclops is placed in 0·2 per cent. HCl, however, the cyclops dies, but the contained embryos become active and escape from the dead bodies of their host.

This is probably what happens. The infected crustacean is introduced into the human stomach, the embryos are there liberated, attain puberty, and copulate; the male then dies and the female starts on her explorations.

The disease is most common in natives, but no race, sex, or age, is exempt.

Symptoms.—After the preliminary infection, the development and travels occupy about a year.

There are occasional feelings of burning, itching, or dragging. Sometimes there may be actual pain and inflammatory swelling along the course of the worm.

Usually there is only a small boil-like inflammation at the site of the small hole which the worm has drilled in the derma.

Stimulation by cold douches results in discharge of portions of milky fluid containing the embryos.

Treatment.—The worm cannot be seized and extracted before the uterus is quite empty, as she resists extraction, and rupture might result in serious inflammation and abscesses.

Cold douches should therefore be given for the fifteen or twenty days during which parturition continues, and the worm can then be wound out with a match or small twig. As an alternative, the body of the worm can be injected with 1 in 1,000 perchloride, which kills it, thus allowing of extraction on the following day.

XII

DYSENTERY

Definition.—An inflammation of the large intestine leading to thickening and ulceration, and characterized by the presence of frequent scanty stools with blood and mucus, accompanied by tormina and tenesmus, and frequently by fever.

Geographical Distribution.—Dysentery has been recognized from remote ages, and occurs in all latitudes. The sporadic and epidemic types may be found almost anywhere, including cold climates.

The endemic type, however, is only a disease of tropical and subtropical countries, and is chiefly found in Africa, Syria, Arabia, India, China, Japan, and South America.

Nature of the Disease.—It should be recognized at the outset that dysentery is not due to one specific cause only.

There are three entirely different dysenteries, due to differing causes, differing in their geographical distribution, in their clinical course, in their sequelæ, and in their behaviour to drugs.

These three types are:

1. Sporadic dysentery.
2. Endemic (or amœbic) dysentery.
3. Epidemic (or bacillary) dysentery.

The following table will show at a glance a comparative résumé of the three types:

TYPES OF DYSENTERY.

	Sporadic.	Endemic.	Epidemic.
Occurs	Anywhere	Tropics or subtropics	Anywhere
Cause	1. Mechanical 2. *Balantidium coli* 3. *Bacillus coli communis* 4. Other bacteria	*Entamœba dysenteriæ* (Schaudinn)	1. Shiga-Kruse bacillus 2. Lenz bacillus 3. Flexner-Strong bacillus 4. Harris-Gay bacillus
Associated organisms	*Bacillus coli communis* and others of intestinal flora	*Entamœba coli* *Paramœba hominis*	*Bacillus coli communis*, etc.
Course	Mild, but acute	Acute first, often chronic later Relapses frequent	Acute Often severe Seldom relapses
Fever	Rare	Rare	Usual
Liver abscess	Nil	Complication or sequela in 5 to 50 per cent. of cases	Nil
Treatment	Purgatives	Ipecacuanha a specific	Ipecacuanha useless Salines and antiseptics indicated

I. Sporadic Dysentery.

Etiology.—The following are recognized causes :

1. *Mechanical irritation* from unmasticated or improperly cooked food.
2. *Balantidium* (*Paramœcium*) *coli.*—This is one of the ciliated infusoria, normally a parasite of swine, and is pathognomonic of sewage pollution.
3. *Bacillus coli communis*—Under certain circumstances this bacillus acquires very virulent properties, and may induce a dysenteric colitis.
4. Other bacilli, such as *B. pyocyaneus*, etc.

Symptoms.—The usual symptoms of dysentery supervene on a preliminary diarrhœa. There is anorexia and a dirty tongue. The griping and tenesmus may be severe, but there is no fever, no tendency to chronicity, and the condition is easily amenable to treatment.

Treatment.—Give a dose of castor-oil, and follow with half a dram of mag. sulph. every hour till free purgation is produced.

Rest in bed and milk diet until three days after the mucoid stools have ceased.

Pathology will be, in a modified degree, similar to that of the endemic and epidemic types of the disease.

II. Endemic (Amœbic) Dysentery.

Etiology.—There are three species of intestinal amœbæ found in man :

1. *Entamœba dysenteriæ* (Schaudinn).
2. *Entamœba coli* (Schaudinn).
3. *Paramœba hominis* (Craig).

Only the first-named is pathogenic, and is the cause of endemic or tropical dysentery.

The following are the morphological characteristics of the *E. dysenteriæ:*

Size, 25 to 35 μ.
Of greenish colour.
Ectosarc is refractile.
Endosarc is coarsely granular.
Contains many vacuoles.
Nucleus (5 μ) lateral and indistinct in fresh specimens.
Actively motile.
Reproduction by spore formation (at periphery of endosarc) and division.

This type of dysentery is endemic in certain parts of the tropics and subtropics, and does not primarily occur in temperate or cold climates.

It is rare in old age and infancy.

One attack predisposes to a recurrence.

Symptoms.—There may be a preliminary diarrhœa for a day or two, after which tenesmus starts, and the stools gradually become mucoid and contain streaks of blood. The symptoms soon become aggravated, and the stools will number from twenty to sixty or more per diem.

After four or five days their character alters to a thin reddish liquid consisting of blood, mucus, pus, and débris, the examination of which will show numerous amœbæ.

The disease may then terminate in one of three ways:

(*a*) *Convalescence*, with or without relapses. This is a slow process. Liver abscess is an occasional sequela, especially if relapses have occurred.

(*b*) *Aggravation*, with gangrene. The tenesmus increases. Sloughs are passed. The motions are offensive. There is a small pulse, cold clammy skin, and great prostration. A typhoid state is often set up. Death from exhaustion is the most frequent result. Occasionally the condition may improve somewhat, and become chronic.

(*c*) *Chronicity*, which may last for years, with pain, emaciation, and much misery.

Treatment.—For this type of dysentery ipecacuanha is a specific.

In *acute cases* see to absolute rest, milk diet, stimulants, hot fomentations to abdomen, hot bottle to perineum.

Powdered ipecacuanha, gr. xxx., should be given twice daily, till the urgent symptoms abate. It is often not well tolerated, and therefore should be preceded (half-hour) by 30 min. of tinct. opii, and a mustard plaster to the epigastrium.

In *chronic cases* the above treatment should be followed as far as circumstances may demand, but for the ipecacuanha may be sustituted:

℞.	Tinct. simarubæ	ʒss.
	Decoct. hæmatoxylin. Decoct. granati cortic.	āāʒii.
	Aq. cinnamon	ad ℥i.

M. Ft. mist.
Sig.: ℥i. every 3 hours.

An enema should also be given twice daily—say:

℞.	Eucalyptol.	1·5.
	Eucalypt. gummi	2·5.
	Aq.	ad 1,500.

Pathology.—In *mild cases* the mucous membrane is congested and softened, with patchy erosions, the submucous tissues infiltrated, and the solitary glands prominent.

In *acute cases* the gut is dilated, with patchy thickening, and ulcers are formed by the sloughing of the solitary glands; while in the aggravated cases there is extensive necrosis with black sloughs.

The liver is hyperæmic and enlarged, and is the seat of abscess in about 4 per cent. of cases.

The mesenteric glands are congested and enlarged.

In *chronic cases* there is contraction of the lumen due to the induration and permanent thickening from fibroid growth in the submucosa.

The liver is sometimes atrophied, and often fatty.

The mesenteric glands are fibrous and pale.

III. Epidemic (Bacillary) Dysentery.

Etiology.—This type of dysentery gives rise to an acute disease, is not followed by liver abscess formation, occurs in epidemics alike in tropical and non-tropical climes, and is attended by fever.

It is due in most cases to a bacillus, the *B. dysenteriæ* (Shiga and Kruse). There are three other allied forms (para-dysentery bacilli) which can all produce the same clinical and morbid symptoms, but in a more modified degree—*i.e.*:

Para-dysentery bacillus I. (Lenz).
„ „ II. (Flexner-Strong).
„ „ III. (Harris-Gay).

The following are the morphological characteristics of the *Shiga bacillus*:

Length, 1 to 3 μ.
Ends rounded.
Non-sporing.
Non-motile.
Decolorized by Gram.
Grows on usual media.
Does not liquefy gelatin.

Symptoms.—The commencement of the disease is more abrupt than in the other types.

The temperature rises several degrees, and may last for several days. The clinical symptoms are otherwise much the same as those mentioned in the previous section. Cases of relapse or chronic cases are, however, rare in this type.

Cases proceeding to aggravated ulceration are common in some epidemics, rare in others.

Occasionally fulminating cases occur, with high temperature and very urgent dysenteric symptoms for three or four days, when the temperature will drop to subnormal, and death from collapse takes place.

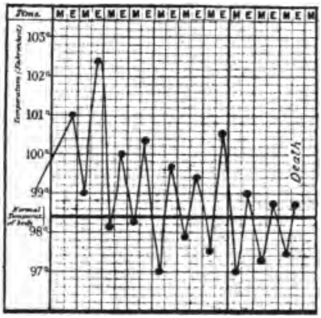

FIG. 7.—EPIDEMIC (BACILLARY) DYSENTERY WITH ADVANCED ULCERATION.

On rare occasions the bacillary toxæmia may cause death almost before the clinical symptoms are manifested.

Treatment.—Absolute rest, milk diet and alcohol will be required, as also hot fomentations to the abdomen.

Drugs will be needed, but ipecacuanha is useless in this type. Salines give the best results.

℞.	Sod. sulphatis	gr. lx.
	Acid. sulph. arom.	♏xx.
	Aq. cinnamon	ad ʒi.

M. Ft. mist.

Sig.: ʒi. every 2 hours till relief is obtained.

Enemata of olive-oil will be grateful; and, if obtainable, anti-dysenteric serum should be given (made from the proper type).

Pathology.—The morbid conditions cited in the section on amœbic dysentery will equally apply in this type.

XIII

EPIDEMIC DROPSY

Definition.—An epidemic disease of the tropics characterized by fever, œdema of the extremities, and usually also a roseolar or purpuric rash, and subsequent anæmia.

Synonyms.—Acute œdema ; acute anæmic dropsy.

Geographical Distribution.—Outbreaks have been reported from Calcutta, Dacca (Bengal), Shillong (Assam), Madras, Mauritius.

Etiology.—The specific cause is unknown.

Children as well as adults are susceptible.

Symptoms.—After an incubation period of a week, fever (without rigor) is usually the first symptom ; it is of a low, remittent type, and lasts either for a few days or, more rarely, for a week or two.

There is occasional diarrhœa.

Dropsy occurs soon after the appearance of the fever, and is first noticed in feet and legs, but may extend to thighs, hands, arms, and more rarely the face. The pleuræ and pericardium may share in the dropsy, which continues for about six weeks, or sometimes longer.

A rash usually accompanies, or soon follows, the œdema. It affects the dropsical parts, and is roseolar at first, becoming purpuric at a later stage, and coalescing to form blotches or streaks.

There is no albuminuria, and the pulse is low tension throughout.

There is no anæsthesia, or loss of knee-jerks.

After a few weeks an anæmic condition is almost always found, the red corpuscles and hæmoglobin being diminished, and the white corpuscles slightly increased.

Death from syncope may occur at about the fifth or sixth day, but the mortality is only 2 to 3 per cent., although higher in some epidemics.

Treatment.—There is no specific drug. Rest, diaphoretics and cardiac tonics are indicated.

Pathology.—The heart may be dilated. Liver enlargement will be only secondary to the heart symptoms.

The spleen is unaffected. The lymphatic glands are normal.

In bad cases the lungs will be found to be much congested.

XIV

FILARIASIS

Definition.—An infection of human beings, mammals, birds, etc., by various species of the genus *Filaria* (other than by *F. medinensis*).

Etiology.—These parasites belong to—

Order—Nematoda.
Family—Filaridæ.
Genus—Filaria.

Their general *morphology* is as follows:

Long, slender, and of uniform breadth.
Anterior extremity rounded.
Males: Smaller than females.
Incurved tail.
Thickened cuticle.
Pre-anal papillæ, 4.
Post-anal papillæ } Variable number.
Spicules }
Females: Vulva opens near mouth.

Man is the definitive host of human filariæ, and certain culicidæ are the intermediary hosts. Various mammalia and birds are the definitive hosts of other filarial species; and fleas, ticks and lice act probably as intermediate hosts in these cases, as well as mosquitoes.

The *history* of the condition may be briefly given.

Pathological results, such as elephantiasis, chyluria, etc., have been known for centuries.

Embryos were first found in 1863, by *Demarquay*, in hydrocele fluid.

They were first found in the peripheral blood by *Lewis*, in Calcutta, in 1868.

The periodicity of certain embryos was demonstrated by *Manson*.

Their extra-corporeal life-history in mosquito hosts was demonstrated by *Manson*, *Bancroft*, *Low*, and others.

As far as is known, only *F. bancrofti* is of pathological significance to man. This parasite and its embryo will alone be considered in this section.

On the adjoining table will be found the practical data of the other chief human filariæ.

Filaria Bancrofti.

This, the only pathogenic filaria of man, was first found by *Bancroft* of Brisbane, in 1876, and the embryo (*Microfilaria nocturna*) by *Demarquay*, in Paris, in 1863, in hydrocele fluid, and by *Lewis*, in Calcutta, in 1868, in peripheral blood.

The *geographical distribution* extends to almost every tropical and subtropical country.

The embryos exhibit a nocturnal periodicity, and only appear in the peripheral blood from about 7 p.m. to 7 a.m.

During the day they retire to the lungs and larger bloodvessels.

The object of this procedure is doubtless to secure their removal from the body by night-feeding mosquitoes.

If the sleep period is changed to the daytime, the periodicity is, however, inverted.

Further *development* takes place in the mosquito.

HUMAN FILARIÆ.

Parental forms ...	*F. loa*	*F. perstans*	*F. ozzardi*	*F. demarquaii*	*F. magalhäesi*	*F. philippinensis*	*F. bancrofti*
Pathogenicity ...	Nil	Nil	Nil	Nil	Probable	Adult not known	Very marked
Endemic area ...	West Africa	West Africa Demerara	British Guiana	West Indies British Guiana	Brazil		Most of tropics and sub-tropics
Site of predilection of parental forms ...	Connective tissues	Post-abdominal and thoracic connective tissues	Subperitoneal connective tissues	Omental tissues	Left ventricle of heart		Lymphatic trunks and varices
Length... ...	M. 25-30 mm. F. 30-40 mm.	M. 45 mm. F. 70-80 mm.	M. 80 mm. F. 85-150 mm.	M. (unknown) F. 85 mm.	M. 83 mm. F. 155 mm.		M. 80 mm. F. 85-150 mm.
Thickness ...	M. 0·3 mm. F. 0·57 mm.	M. 0·12 mm. F. 0·25 mm.	M. 0·21 mm. F. 0·3 mm.	M. — F. 0·32 mm.	M. 0·4 mm. F. 0·7 mm.		M. 0·2 mm. F. 0·26 mm.
Head to							
Vaginal outlet	—	0·6 mm.	0·71 mm.	0·6 mm.	2·56 mm.		0·71 mm.
Ovarian opening	—	?	0·85 mm.	—	—		0·92 mm.
Tail to anal papilla... ...	—	0·145 mm.	0·230 mm.	0·25 mm.	0·13 mm.		0·225 mm.
Nature of tail ...	Incurved, with two terminal tubercles	Bulbous, incurved, and mitred	Bulbous Cuticle not thickened	—	Rounded		Blunt, circular, not bulbous

Name of embryo ...	*Microfilaria diurna*	*M. perstans*	*M. ozzardi*	*M. demarquaii*	Unknown	*M. philippinensis*	*M. nocturna*
Length of embryo	0·3 mm.	0·2 mm.	0·21 mm.	0·2 mm.	Embryo not known	0·32 mm.	0·3 mm.
Breadth of embryo	0·0075 mm.	0·0045 mm.	0·005 mm.	0·005 mm.		0·0065 mm.	0·0075 mm.
Sheath of embryo	Present Loose	Absent	Absent	Absent		Present Tight	Present Loose
Head of embryo ...	Six lips	Papillated	—	Spine		Serrated retractile band	Six lips
Tail of embryo ...	Pointed	Blunt	Pointed	Pointed		Pointed and abruptly attenuated	Pointed
Anterior V. spot of embryo ...	Present	—	—	Present		Present	Present
Viscus of embryo	Granular mass	—	—	—		A spiral tubular cylinder	Granular mass
Posterior V. spot of embryo ...	Present	—	—	—		Present Also a papilla	Present
Motility of embryo	Lashing Not progressive	Lashing and progressive	? Progressive	Progressive		Lashing and progressive	Lashing Not progressive
Periodicity of embryo	Diurnal	Nil	Nil	Nil		Nil	Nocturnal
Intermediate mosquito hosts ...	? *Chrysops dimidiatus* (the mangrove fly) Or possibly mosquitoes	? The tick *Argas monbata* Mosquitoes so far not successful	—	? *Stegomyia fasciata*	—		*Culex fatigans* *Myzomyia rossii* *Pyratophorus costalis* *Panoplites africanus* *Stegomyia fasciata*

The filarial embryo passes into the mosquito's stomach, breaks through its sheath, pierces the stomach wall, becomes quiescent for about a week, by which time it is 1·5 millimetres long and has a mouth and anus.

It then becomes active and works through the mosquito's tissues till it reaches the salivary glands, and thence passes into a new human host through the mosquito's proboscis when in the act of biting.

Soon it finds its way to the lymphatic trunks, where copulation occurs, and a stream of embryos subsequently enters the circulation.

Prophylaxis will therefore consist of—

1. Isolation of infected.
2. Use of mosquito-nets.
3. Anti-mosquito campaigns.

Symptoms, Treatment, and Pathology.—It is to the parent filariæ that the pathogenic symptoms are due. These are the sequelæ of mechanical obstruction in the lymph vessels.

1. **Lymphangitis.**—There is very frequently a lymphangitis, especially in cases of elephantiasis, along the course of obstructed lymphatics. This is accompanied by rigors and fever (elephantoid fever). Thickened tissues remain after the swelling has subsided.

Rest, elevation, and local anodynes form the treatment.

2. **Varicose Glands.**—A slow and painless enlargement of the glands in one or both groins. They are soft to the touch, and the skin moves freely over them.

Chyluria may also be present. A diagnosis of hernia should be excluded, for the tumour is not tympanitic, nor is there an impulse on coughing.

Treatment is inadvisable.

3. **Elephantiasis.** — An inflammatory lymphatic œdema which may attack many parts of the body. In 95 per cent. of the cases the lower limb is the one affected. More rarely the arms, mammæ, vulvæ, or scrotum, may be involved.

A lymphangitis will begin the disease, often accompanied by a diffuse cellulitis and fever.

Recurring attacks leave a permanent thickening of the tissues, until the affected part may reach an enormous size. This swelling feels brawny, and does not 'pit' much on pressure.

The treatment will usually be surgical.

If seen very early, non-inflammatory cases may be relieved by elevation and pressure.

The mammæ, scrotum, or vulvæ, may be removed if the tumour is inconveniently large.

4. **Lymph Scrotum.**—Lymphatic varices give rise to an increasing hypertrophy of the scrotum. Sinuses will often form, discharging a milky fluid in which embryos are found in great numbers. Small abscesses are a frequent accompaniment.

Treatment is on surgical lines. Amputation is sometimes followed by elephantiasis of the leg or chyluria.

5. **Chyluria.**—A condition due to lymphatic varices in the bladder wall. These rupture, and chyluria and hæmaturia result.

In endemic centres of bilharzia, that disease may have to be excluded.

The condition is serious, but not directly fatal.

Drugs do not appear to have much effect.

Rest in bed, with elevation of the pelvis, and laxatives, are indicated.

XV

FRAMBŒSIA

Definition.—A tropical contagious disease of a chronic type, characterized by a fungating eruption, and tending to a spontaneous cure.

Synonyms.—Yaws; pian; parangi.

Geographical Distribution.—The first mention of the disease is by *Oviedo*, in San Domingo, in 1525.

It is found in Malaya, Assam, Burma, Siam, India, Ceylon, China, Fiji, and is especially endemic in West Africa, the West Indies, and South America.

Etiology.—The usual etiological gamut of cocci, bacilli, and moulds has been played through with variations, but without striking the right note.

The condition is probably a spirochæte infection. *Castellani* found these in smears, and they were pronounced by *Schaudinn* to comprise three species:

S. refringens (Schaudinn).
S. tenuis (Castellani).
S. pallidula (Castellani).

No race or age is exempt, but it is more especially a disease of the children of dark-skinned races.

One attack confers immunity. It is highly contagious.

It has many resemblances to syphilis, but is neither congenital nor hereditary, nor is it conveyed by suckling. The hair does not fall out, and neither viscera nor nervous system are attacked.

Symptoms.—After an incubation period of twelve to twenty days, the disease, which is contracted through a cut or skin abrasion, exhibits a small ulcer at the site of inoculation. This may heal, or become inflamed. There is slight constitutional disturbance—pyrexia, headaches, dyspepsia, etc.—and after five or six weeks from the original infection a general eruption occurs. This consists of papules up to the size of a pea, and is most frequently found on exposed parts.

The papules develop a cheesy core, and exude a serous moisture which forms scabs. They attain maximum development in two weeks. There is considerable itching, but they are not sensitive to acids.

As a rule, after a few weeks' life they shrink, and eventually leave pigmented cicatrices, but in 8 per cent. of cases they break down and ulcerate, leading to necroses, periostitis, extensive contractions, etc.

The case mortality is about 2·5 per cent.

Treatment.—Potassium iodide and mercury are of value internally.

The ulcers should be treated antiseptically.

The food should be nourishing.

The sick should be isolated, and infected quarters suitably disinfected.

Pathology. — It is a chronic dermatitis. The papillæ are elongated and their vessels dilated.

The sweat glands are dilated.

The hair follicles are unaffected.

There is a cellular infiltration of plasma cells, an extravasation of polymorphonuclear leucocytes, and also a hyperkeratosis.

XVI

GLANDULA IDIOPATHICA

Definition.—An idiopathic lymphadenitis occurring both in tropical and temperate climates, of neither venereal nor plague origin, and usually accompanied by slight febrile symptoms.

Synonyms.—Climatic bubo; non-venereal bubo.

Geographical Distribution.—It is fairly common in the British Navy, the greatest number of cases occurring on the tropical stations.

Cases are reported from India, Hong-Kong, Japan, Madagascar, German East Africa, Sumatra, and Malta.

Etiology.—The disease is one in which a venereal origin can be excluded.

No plague bacilli are found, and, moreover, cases occur in temperate climates amongst men who have not been exposed to plague in any form; a plague origin can therefore be excluded.

That it occurs both in temperate and tropical climates will negative climate as a causal factor.

A bacterial origin has been claimed, but not substantiated.

Young males are the usual sufferers.

Symptoms.—Malaise and headache may occur as prodromata.

Then a febrile attack begins, being irregular and remittent, and lasting from one to two weeks, or more.

The lymphatic glands enlarge soon after the fever begins.

This enlargement is usually unilateral and inguinal. It progresses rapidly, and may attain the size of a goose's egg. It is indurated and painful. In sixty per cent. of cases, the glands are absorbed, and forty per cent. suppurate.

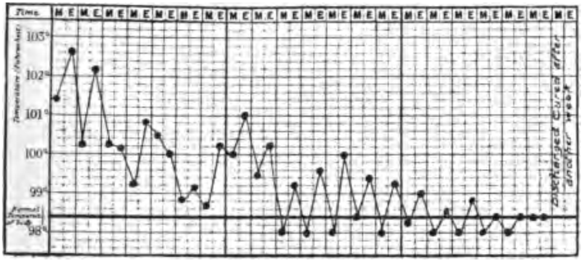

FIG. 8.—TEMPERATURE IN GLANDULA IDIOPATHICA.

Treatment.—A preliminary purge; paint the bubo with iodine and incise if suppuration occurs; give 2 grains of calcium iodide thrice daily; alcohol may be given if the patient is used to it; a tonic during convalescence is advisable.

XVII

GOUNDOU

Definition.—A bony tumour affecting the nasal processes of the superior maxillæ.

Distribution.—Limited to West Africa.

Etiology.—The exciting cause is not known, but apparently it is not hereditary.

A connexion with yaws, and also with the deposit of dipterous larvæ, has been suggested, but not substantiated.

It is more common in men than women, and has been noted in monkeys.

Symptoms.—Soon after childhood a persistent frontal headache is complained of, and is accompanied by a nasal sanguino-purulent discharge. At the same time there is symmetrical enlargement of the nasal processes.

The tumours increase in size, while the other symptoms subside.

Treatment.—The removal of the tumour is alone efficacious.

Pathology.—The tumour consists of thin compact bone, with a cancellous core, the skin above being normal and movable.

XVIII

HEPATITIS

Definition.—An inflammatory condition of the liver.

Etiology.—The liver of the tropical resident is liable to the same quota of disease as is that of the resident in cooler climes.

Life in the tropics, however, adds an extra burden to the liver.

1. High temperature at first stimulates, and later diminishes, liver activity.
2. The liver has more work to do, because the CO_2 output from the lungs is lessened.
3. The liver shares in the general impairment of nutrition and impoverished blood.
4. Dietetic errors are frequent.
5. An excessive amount of alcohol is taken.
6. Dysentery and malaria have an important bearing on the liver.
7. A sudden chill or over-exposure to the sun may convert a congested liver into an acute hepatitis.

Symptoms.—A *congested liver* will mean—Headaches, dirty tongue, diarrhœa, hepatic fullness or aching, heavy sleep, and often fever.

Continued or frequently repeated attacks, such as the above, may either lead to an acute hepatitis, or else to the well-known *cachexia tropica*, made so familiar to us in the person of the retired Anglo-

Indian, with his sallow complexion, irritable temper, listlessness, bad digestion, and constipation.

If, however, the graver condition supervenes, and an *acute hepatitis* occurs, it will be ushered in by rigors, and include a sharp attack of fever, an enlarged liver with pain and tenderness, pain in the right shoulder and down the arm, furred tongue, anorexia, jaundice, constipation, clay-coloured stools, depression, hurried respiration, etc.

Treatment.—

1. *For an Early Hyperæmia and Congestion:*

(*a*) Rest and light diet.
(*b*) Mustard plaster to hepatic region.
(*c*) Preliminary purge:

℞.	Extr. colocynth. co.	gr. i.ss.
	Hyd. subchlor. Extr. jalap.	āā gr. i.

M. Ft. pil. Sig.: Pil. i. statim.

(*d*) Prescribe 1 ounce of each of the following, to be mixed, and drunk during effervescence, shortly before food:

℞.	Sod. bicarb.	ʒiii.
	Ammon. carb.	ʒss.
	Aq.	ad ℥vi.

℞.	Acid. cit.	ʒi.ss.
	Syr. limon.	ʒvi.
	Aq.	ad ℥vi.

2. *For an Acute Hepatitis:*

(*a*) 5 grains of calomel.
(*b*) Rest in bed and milk diet only.
(*c*) Hot bath and bed and blankets.

(*d*) Poultice over whole liver twice daily till pain and fever have gone.

(*e*) ℞.	Ammon. chlor.	gr. xx.
	Extr. cascar. liq.	♏ xxx.
	Aq.	ad ℥i.

Sig.: ℥i. t. d. s. till pain and fever have gone.

XIX

INFECTIOUS JAUNDICE

Definition.—An acute infectious disease characterized by fever, jaundice, enlarged liver and spleen, nephritis, and nervous symptoms.

Synonyms.—Febrile jaundice; epidemic jaundice; septic jaundice; Weil's disease; Bulgarian fever.

Geographical Distribution.—Small epidemics have occurred in England, the United States, and North China.

A more extended epidemic visited the Balkans during the Russo-Turkish War.

The disease is endemic in parts of Turkey in Asia.

Sporadic cases are reported from Egypt, Malta, Greece, and Turkey.

History.—Professor *Weil* of Heidelberg called attention to the condition in 1886. The disease is probably identical with the thirty-four epidemics reported by *Hirsch* as occurring in Europe between 1745 and 1885.

Etiology.—The incidence is most frequent in males between twenty and thirty years of age.

The specific cause has not been discovered.

There would seem to be some connexion between the disease and the presence of putrid animal matter and sewage.

The disease is not contagious, but often highly infectious.

Culex fatigans or *Stegomyia fasciata* may possibly be a transmitting agent.

Symptoms.—There is an incubation period of one or two days, after which headache, vomiting, and lumbar pain usher in the disease.

There is often a rigor, and the temperature rises quickly four or five degrees.

After three to five days the fever subsides, and is followed by jaundice, which lasts for seven to nine days, and is accompanied by enlargement of spleen and liver, with albuminuria.

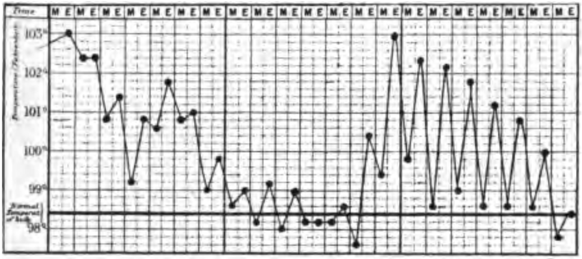

FIG. 9.—TEMPERATURE IN INFECTIOUS JAUNDICE.

A secondary rise of temperature ensues, and lasts for a week and more.

The stools become clay-coloured during the stage of jaundice, which usually disappears at the beginning of convalescence.

The urine is acid throughout, is scanty, contains albumin, and the urea is diminished. During the second stage bile pigment is present, and blood-casts may be found. On the seventh to ninth day a urinary crisis occurs, when the secretion is re-established, with diminished albumin and increased urea.

Hyperpyrexia and typhoidal symptoms may occur during the course of the disease.

In fatal cases death usually occurs at about the end of the first week, from uræmia or syncope.

The mortality varies from 10 to 60 per cent., according to the virulence of the outbreak.

Treatment.—Calomel and quinine have been advocated, but are not of much use.

Some intestinal antiseptic may be tried.

The diet should be limited to milk during the acute stages. Plentiful water is indicated, and heart symptoms should be treated as they arise.

Pathology.—The liver is enlarged, and shows early fatty degeneration, with small-celled infiltration of the portal canals.

The spleen is also enlarged, with pin-head yellow spots scattered throughout the pulp.

The kidneys are large and congested, with infiltration round the glomeruli.

XX

KALA-AZAR

Definition.—A chronic infective disease of the tropics characterized by irregular pyrexia and splenic enlargement, leading to a cachectic condition of emaciation and anæmia.

Synonyms.—Black fever; Dum-dum fever; tropical febrile splenomegaly.

Geographical Distribution.—The disease has not been much investigated up to the present, but probably has a wide tropical and subtropical distribution.

Assam has suffered epidemically since 1875, and has now become a permanent endemic focus. There are several other endemic foci in India, as at Dum-dum and Burdwan.

Sporadic cases have been reported from Hankow Pekin, Manila, Penang, Algiers, the Soudan, etc.

Etiology.—The disease is due to a parasite, the *Leishmania donovani*, which is present in the spleen of every case of kala-azar, and in the peripheral blood of advanced cases.

Leishman found these parasites in post-mortem spleen smears of a military patient from India, who died at Netley, and he, in 1903, was the first to describe them.

The *Leishmania donovani* is one of the flagellated protozoa, but belongs neither to the piroplasmata nor trypanosomata.

The following are the chief *morphological details:*

1. They are oval or circular, and from 2·5 to 3·5 μ in size.

2. There is a large chromatin mass at the periphery of the parasite, which stains only lightly with Romanowsky.

3. A smaller chromatin mass, staining deeply, is found opposite the larger one.

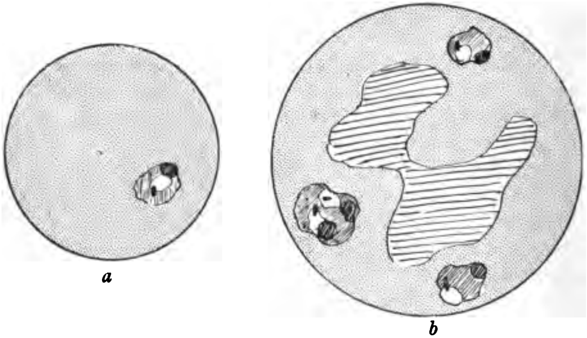

FIG. 10.—*LEISHMANIA DONOVANI:*

a. A parasite in red blood-corpuscle.
b. Three parasites in polymorphonuclear leucocyte (one dividing).

4. There are usually one or two vacuoles displacing the cytoplasm.

5. Most of the parasites in splenic smears are within the large hyaline cells (macrophages); a fewer number are found in endothelial cells; less still in large mononuclears; and least in the polymorphonuclear leucocytes, or free in the blood.

6. Development is by division. The large chromatin mass divides first.

7. Further development can be observed by mixing the spleen pulp with acid sodium citrate solution. Flagella appear, and further subdivision ensues.

8. Since the parasite in this disease gives rise to malignant conditions, while that in granuloma endemica is more benign and of different geographical distribution, it is probable that they are different species.

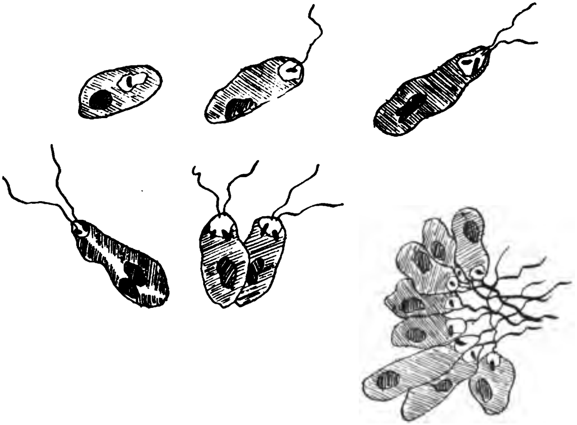

FIG. 11.—*LEISHMANIA DONOVANI:*

Showing formation of Flagella and Division of Flagellated Forms.

With regard to its *epidemiology*, no race, sex, or age is immune.

The incidence is greatest amongst acclimatized residents.

Epidemics can usually be traced to an imported case. People in that house are usually the first to suffer.

The house retains infection for many months.

Its method of spread favours the probability of a *cimex*, rather than a flea or mosquito, being the intermediate host. The parasite can develop into the flagellate stage in the bed-bug's stomach.

Symptoms.—There are seldom any prodromal symptoms.

A febrile attack comes on, and lasts for two or three weeks. It is remittent and often severe, and is quite uninfluenced by quinine. At the same time the spleen and liver become enlarged.

Apyrexia and general improvement follow, but only for a short time. Continued attacks and remissions alternate until a cachexia is established, in which there is a continuous low fever, progressive enlargement of the spleen, anæmia, and much emaciation.

Other *complications* are sometimes found, such as phagedænic processes, purpura, epistaxis, œdema of the feet, diarrhœa, dysenteric symptoms, and, more rarely, skin pigmentation.

The *blood changes* are important.

The absolute leucocytic count falls to between 1,000 and 2,000 per cubic millimetre, which is lower than in other fevers.

Relatively, there is an increase of large mononuclears, which, in the early remittent fever, is a most useful diagnostic point in excluding typhoid.

There is also, relatively, a decrease of polymorphonuclears, which will predispose to the secondary bacterial invasions which are usually the last straw; experience has also shown that, as they become fewer, the prognosis grows worse.

The *case mortality* is about 96 per cent.

Treatment.—This is rather unsatisfactory. There is no specific.

Rogers strongly recommends very large doses of quinine, claiming to convert a high remittent fever into a less harmful, low, intermittent type, with a consequent reduction of mortality from 96 to 76 per cent.

Bone-marrow tabloids should be given for blood repair.

Atoxyl, as used in trypanosomiasis (q.v.), might with advantage be tried.

Pathology.—The *spleen* is much enlarged, hard, and firm, but is friable and has no fibroid changes.

It retains its shape after removal from the body.

Microscopically, immense numbers of parasites will be found in the smears.

The *liver* is firm, but friable. In the more chronic cases there is actual intracellular cirrhosis, and the liver cells show extreme atrophy.

The *large intestine* frequently exhibits extensive ulceration.

Cancrum oris and other septic conditions, such as mastoid abscess, otitis media, etc., are most frequently found as complications.

Pneumonia is the next most frequent and fatal complication.

XXI

LATAH

Definition.—A cerebral neurosis manifested by involuntary movements or sounds, and induced by fright or suggestion.

Distribution.—Chiefly limited to the Malay Peninsula and Archipelago, and to a less extent found also in Burmah and Siam.

Etiology.—This is very obscure. There is apparently no connexion with hysteria, epilepsy, or other common nervous disorders.

During the attacks the intellect is unimpaired, and consciousness is maintained.

It is rather more common in young women than in men, and would appear to be incurable.

Excessive venery is a possible predisposing cause.

Symptoms.—An attack may be excited by fright, or even merely by demonstrative movements.

Actions are involuntarily mimicked (echokinesia); words may be involuntarily repeated (echolalia); and orders given are executed even though ludicrous or repulsive.

Treatment.—This is apparently of little avail, and it is therefore fortunate that the condition is not a serious one.

XXII

LEPROSY

Definition.—A chronic disease characterized by the formation of neoplasms giving rise to nodosities or perversions of sensation, and to progressive mutilations.

Synonym.—Elephantiasis Græcorum.

Geographical Distribution.—The disease is of remote antiquity, and has been recognized for many centuries in Egypt, India, and Japan.

At one period it was very prevalent in Europe. It gradually disappeared, leaving England before the time of Henry VIII.; the last case in Scotland was reported from the Shetland Islands in 1798.

The only remaining European endemic centres are Scandinavia and Iceland.

It is widely spread throughout the tropics and subtropics of Asia, Africa, America, and the West Indies.

Etiology.—The specific cause of leprosy is a bacillus —the *B. lepræ*—discovered by *Hansen* in 1871.

Its *morphology* is as follows:

Slender rods 5 μ in length.
Stains with usual stains.
Stained by Gram.
Non-motile.
Non-liquefying.
Does not form spores.
Is hard to cultivate, but has been grown on a

nutrient glucose agar, growth ceasing after twenty days, and proving impossible to renew by subculture.

Found in all leprous tissues and discharges.

Exists both in cells and free.

With regard to the *incidence*, the disease is more common amongst males, and most frequently begins between the ages of fifteen and thirty.

Neither race, temperature, nor altitude appear to have any determining influence, and the channels of infection are at present unknown.

The following theories have all had support:

1. Transmission by contagion.
2. ,, ,, the air.
3. ,, ,, heredity.
4. ,, ,, diet.
5. ,, ,, intermediate hosts.

None of these theories have been proved, and the first four, at any rate, are highly improbable.

Of the intermediate hosts we may neglect ticks, lice, and flies.

The *B. lepræ* has been demonstrated in a mosquito (*C. pungens*), but the distribution of the disease does not coincide with that of any known species of mosquito.

Fleas might be thought of, since a rat disease has been found, in which the bacteria are morphologically and culturally identical with *B. lepræ*.

The greatest probability is in the direction of the bed bug (*Cimex lectularius*), for the following reasons:

1. The bacillus has been found in it.
2. The distribution of *Cimex* is world-wide.
3. Segregation of lepers and improved sanitary principles throughout Europe, by diminishing the numbers as well as the infected diet of the

bugs, would account for the gradual disappearance of the disease from that Continent.

4. No other intermediate hosts would account for the immunity of nurses and officers in leper asylums.

The following are the *Conclusions of the Indian Leprosy Commission*, 1890 :

1. Leprosy is a disease *sui generis*, and not a manifestation of syphilis or tuberculosis.
2. It is not hereditary.
3. It must be regarded as contagious and inoculable.
4. It is not originated by food, climate, or insanitary surroundings, but these causes may predispose to the disease.
5. The method of origination is unknown.

Symptoms.—The *incubation period* may extend from four weeks to four years, or even longer.

Dyspeptic troubles then begin, often accompanied by recurring attacks of fever, much debility, hyperæsthesia, and hyperidrosis.

A little later erythematous or pigmented maculæ appear on the face, extensor surfaces of the limbs, back, chest, or abdomen. These eventually become permanent and anæsthetic, and the hair of the affected region falls out.

Leprosy is now established, and may subsequently develop either as a condition in which nodular elements predominate (*lepra tuberosa*), or anæsthetic elements (*lepra maculo-anæsthetica*).

1. *Lepra Tuberosa.*—Infiltrated patches or hard, firm nodules appear, and the resulting deformity of the face is known as *facies leonina*, from the fierce expression caused by the heavy and furrowed infiltration of forehead and cheeks. The lobules of the ears are usually affected also.

The infiltration extends to the eyes, affecting the cornea, and later the ciliary nerves, choroid, and retina.

The soft parts of the nose become softened, ulcerate, and disappear.

The tongue, pharynx, and epiglottis may all be involved.

A non-suppurating lymphadenitis is common.

The testicle, spleen, and liver are always affected.

In addition to these morbid processes, one or more nerves may share in the disease, giving rise to early pain and late anæsthesia.

Notwithstanding this sombre picture, the general health of the patient remains remarkably good, and death eventually is not due directly to the leprosy, but rather to (*a*) amyloid degenerations following extensive ulcerations; (*b*) nephritis; or (*c*) tuberculosis.

The prognosis is wellnigh hopeless, and the duration of life is usually eight to nine years.

2. *Lepra Maculo-anæsthetica.* — In this type the nervous system, rather than the other body tissues, is involved.

Serpiginous ulceration may follow bullæ. The early implication of the nerves leads to neuralgias and severe hyperæsthesia, and subsequently to paresis and muscular atrophy.

Owing to this paralysis, the facial expression is lost and the eyes cannot be closed. The hands become claw-like (*main-en-griffe*).

Trophic changes result in necrosis of feet and hands, so that toes and fingers, and even the whole foot, may disappear.

After some years the bacillary activity very often ceases, leaving only its after-affect—anæsthesia.

This type is not nearly so serious as the nodular type, and is essentially chronic, lasting even up to forty years, although death may take place earlier from intercurrent disease.

Treatment.—The most usual, and perhaps most encouraging, drug is *Chaulmoogra-oil*—the oil of the nut *Gynocardia odorata.*

This oil is rubbed twice daily for a quarter of an hour over the affected areas; and at the same time it is given in capsules by the month, beginning in doses of 10 minims three times daily, and gradually increasing it up to a daily maximum of 2 drams. This should be persisted in honestly for several years, and at the same time attention to the general health should be secured by fresh air, exercise, nutritious food, and occasional tonics.

Large quantities of sodium chloride should be included in the diet, and hot salt baths given occasionally, since salt undoubtedly inhibits the growth of *B. lepræ.*

Leprolin—a glycerine dilution of a filtered culture—may be used for injections. Good results have followed this treatment in India.

The various lesions of leprosy should be treated on the usual surgical lines as the occasion may demand.

Pathology.—The *nerves* most frequently implicated are the facial, median, radial, ulnar, posterior tibial, and peroneal. The perineurium is infiltrated with lepra cells, which later spread to the endoneurium and cause degeneration.

The *glands* are much infiltrated, and, in old cases, sclerosed. Caseation and suppuration do not occur.

The *testes* are infiltrated and sclerosed.

The *liver* is normal in appearance, but microscopically shows interlobular infiltration.

The *spleen* is apparently normal, but shows many lepra cells.

The *leproma* is smooth and glistening on section, and contains lepra cells (nucleated cells, about the size of polymorphonuclears), some large epithelioid cells, fragments of connective tissue, and bloodvessels. The sweat and sebaceous glands and hair follicles, in the infiltrated area, disappear, and the corium becomes fibroid.

Bacilli occur both free and in the lepra cells.

XXIII

LIVER ABSCESS

Definition.—A single or multiple abscess formation in the liver, connected with residence in the tropics or subtropics, and associated with amœbic dysentery.

Geographical Distribution.—This follows the distribution of endemic (amœbic) dysentery, and is found more especially in Egypt, Syria, Arabia, India, Malaya, China, Japan, and some parts of South America.

Etiology.—The *predisposing causes* are—

1. Amœbic dysentery, which must be present or have previously occurred in all cases.
2. The male sex, contributing 95 per cent. of cases.
3. Recent tropical residence is noted in 50 per cent. of cases.
4. Middle age, during which nearly every case occurs.
5. European nationality, for only 3 per cent. of cases are amongst natives.
6. Abnormal liver conditions, such as may be caused by malaria, over-eating, or over-drinking.

The *specific cause* is the *Amœba coli*, either alone or with other organisms. It is identical with the amœba found in the intestine of endemic dysentery, and is invariably present at an early stage in the contents or walls of the liver abscess.

In multiple abscesses there are usually bacteria associated with the amœba, such as *S. pyogenes aureus*, *B. coli*, and more rarely *B. pyocyaneus*, and others. Such mixed infection tends to give rise to a different clinical type of disease, with rigors, profuse sweats, and septic temperatures. Later in the disease the abscesses may be sterile.

The *method of infection* is almost certainly from the bowel, via the portal circulation. Rarely, the amœba may pass through continuous tissue.

Symptoms.—These are very varied, and diagnosis is often difficult.

A large abscess may be present with scarcely any signs or symptoms.

The liver is enlarged, usually an upward increase of the right lobe.

There is usually some pain referred to the shoulder.

In simple amœbic infections there is often but little acute pain, although a general soreness and tenderness may be present.

The facial expression is often haggard, and the skin of a greyish-green appearance.

The temperature may remain normal, or fever may be constant. Mixed infections will show a septic temperature, with rigors and profuse sweats.

There is an absolute leucocytosis in all cases.

The *mortality* varies from 60 to 80 per cent.

If the abscess is multiple, and due to a mixed infection, it has a rapid development, and ends in death from septicæmia without perforation.

In multiple abscesses of pure amœbic origin, there is less toxæmia, and perforation not infrequently will occur, usually into the right lung or right pleura, and more rarely into the abdomen or intestine, etc.

Solitary abscesses have a more chronic course, and may continue for a year or more.

In this latter type, if recognized and dealt with, the *prognosis* is not unfavourable; but in multiple cases it is bad, and almost hopeless in septic cases.

Treatment.—This is surgical. If an abscess is suspected, and no local signs are made out, then exploration should be made with a syringe, upwards, inwards, and backwards, in the eighth or ninth interspace in the right axillary line. Several punctures should be made before giving up hope.

A tender spot or local bulging may, however, indicate the site for exploration.

Having found pus, the needle should be left *in situ*, a three-inch incision made, and the liver exposed, if necessary resecting some rib. The liver capsule should be sutured to the abdominal wall if there are no adhesions, and then a Grim's forceps is pushed into the abscess, opened, and a drainage-tube inserted.

An alternative method is Manson's operation, in which, after pus is found, the abscess is reached by a trochar and cannula.

The dressings should be changed twice daily.

If all is going well the temperature will fall to normal.

Imperfect drainage or septic infection will keep the temperature up, and the conditions should be rectified on the usual lines. If the temperature still keeps up, notwithstanding antiseptic douches and drainage, then it is probably due to further abscesses, which should be promptly sought for.

Pathology.—In nearly every case the right lobe is involved, and in 70 per cent. of cases the right lobe only.

In about half the cases the abscess is a solitary one.

The abscesses are generally near the surface, and may vary in size, some being only 1 millimetre in diameter, others occupying a whole lobe.

XXIV

MADURA-FOOT

Definition.—A tropical disease characterized by chronic enlargement of the foot (or occasionally the hand), the formation of sinuses, and inflammatory degeneration.

Geographical Distribution.—The disease is endemic in East Africa, Madura, Delhi, and other Indian districts, especially in Kashmir and the Punjab.

Sporadic cases have been reported from China, North Africa, Madagascar, Cuba, Demarara, Malaya, etc.

History.—*Kämpfer* was the first to call attention to the disease, in 1712. *Carter* first recognized it as a mycosis.

Etiology.—It is chiefly a disease which attacks middle-aged natives.

The specific cause is a schizomycete—*Cladothrix mycetomæ*. This has not been isolated yet from soil or vegetable matter.

Symptoms.—Some abrasion forms the site of inoculation. Painful or painless induration results, and eventually breaks down in about eight weeks to form a fistula, which discharges white or black granules, resembling 'fish-roe.'

The condition is repeated at various foci, until the whole foot is swollen and honeycombed.

The disease may continue for many years, and death occurs from exhaustion or intercurrent disease.

Treatment.—Drugs have but little effect, though calcium iodide might be tried.

The disease is purely local, and should be treated on surgical lines. Excision of the initial lesion if seen early, or amputation at a later stage.

Pathology.—The 'fish-roe' granules are formed by the fungus, the black variety being merely a degeneration form of the white.

The tissues of the affected member, including the bone, all become disintegrated. Cheesy masses of granules fill the cysts and sinuses.

XXV

MALARIA

Definition.—A specific protozoal infection of the blood by several species of the genus *Hæmamœba*, giving rise to febrile paroxysms amenable to quinine.

Geographical Distribution.—Malaria is widespread through temperate and tropic countries.

In temperate regions the type of disease is benign. It is not seen above the latitude of 60° N.

It is endemic in most parts of the tropics and subtropics, but is not known in most of the Pacific islands or Australia, and in Japan it is rare.

Etiology.—The parasites which cause the disease belong to the—

Kingdom—Protozoa.
Class—Sporozoa.
Order—Hæmosporidia.
Genus—Hæmamœba (Grassi).

There are a fair number of species in this genus, some of which are confined to monkeys, birds, etc., and give rise to pathogenic symptoms akin to human malaria.

In human malaria there are at least three incriminated species, and possibly more ; these are :

H. vivax (the parasite of 'benign tertian').
H. malariæ (quartan fever).
H. præcox (malignant tertian).

These parasites were first found in human blood corpuscles, by *Laveran*, in 1880, in Algiers.

For some time subsequently it was not known by what means the parasite could continue its life-history outside the human host. *King*, in 1883, suggested mosquitoes as possible intermediate hosts. This theory received absolute confirmation by the experiments of *Ross* (1897-99), who traced an exogenous life-cycle within the mosquito's body.

Not *all* mosquitoes, however, prove to be efficient hosts. The following list comprises those species with regard to which positive proof has, up to the present, been established:

Anopheles maculipennis (Europe, North America, and Africa).
Anopheles bifurcatus (Europe).
Anopheles jesoensis (Japan).
Anopheles algeriensis (Africa).
Cellia albipes (West Indies).
Myzorhynchus pseudopictus (Europe).
Myzorhynchus paludis (Africa).
Myzomyia hispaniola (Africa).
Myzomyia funesta (Africa).
Myzomyia listoni (India).
Myzomyia culicifacies (India).
Nyssorhynchus lutzi (Central and South America).
Nyssorhynchus cubensis (Central and South America).
Nyssorhynchus maculatus (India).
Pyretophorus superfictus (Europe and Africa).
Pyretophorus costalis (Africa).

In addition to the finding of the parasite, and the demonstration of an intermediate life-history in the mosquito, an *experimental proof* was established by experiments carried out in 1900 on behalf of the

Colonial Office and the London School of Tropical Medicine.

A select party spent the three most malarial months of the year in the worst districts of the Roman Campagna—the only precautions taken being the use of mosquito-proof huts. Malaria was rife in the surrounding districts, but the experimental party were completely immune.

While this was proceeding, mosquitoes which had been fed on malarial blood were dispatched from Rome to London, and allowed there to bite two Europeans who had never left England, nor had been exposed to any malarial influences. Both these persons developed typical malaria in about fourteen days, and the parasites were duly found in their blood.

Predisposing causes will consist of the proximity of infected natives, and any atmospheric or local conditions which will favour the breeding of susceptible mosquitoes. The latter cause will be contributed to by any public works, such as railway cuttings, etc., which cause a rise in the subsoil water.

With regard to the *incidence* of the disease, certain natives in endemic areas seem to exhibit some immunity. Otherwise all races are equally liable to attack, children and infants being more susceptible than adults.

The *percentage of infected children under ten years of age* is an index to the amount of malaria in any district; this figure is called the *Endemic Index of Koch.*

Life-History of the Parasite.

The malarial parasites have two methods of reproduction :

1. *Schizogony* (σχίζω, to cleave), which is an endogenous reproduction taking place within the body of its ultimate human or other host; and
2. *Sporogony*, or an exogenous cycle, part of which takes place in the body of a mosquito intermediate host.

Schizogony.—Spindle-shaped spores called *sporozoites* (*zygotoblasts*) are introduced by the bite of an infected mosquito. They each enter a red corpuscle and begin to grow, becoming amœboid *trophozoites*. These gradually fill the whole corpuscle, black grains of melanin being formed in their substance.

When they are full grown they are known as *schizonts*, and as the pigment gradually collects towards the centre, the nucleus divides, the protoplasm segments, until, finally, round spores are matured, which are called *merozoites*, and these break away to attack a new corpuscle each.

The number of merozoites is usually twelve to twenty-four in the case of the benign tertian parasite (*H. vivax*); nine to twelve in quartan infections (*H. malariæ*); and variable but few in the malignant variety (*H. præcox*). These malignant merozoites are sporulated in the liver, spleen, and bone marrow, and not in the peripheral blood, as are those of *H. vivax* and *H. malariæ*.

The endogenous cycle is thus completed, and the increase, being by geometrical progression, is of course rapid.

Sporogony.—The febrile reaction caused by the above process acts as a stimulus for the production of certain sexual forms known as *sporonts* (or *gametocytes*). These are probably formed by a percentage of trophozoites instead of the usual schizont formation. Their early development takes place in the spleen and bone marrow.

After reaching a certain size they circulate in the peripheral blood as male and female sporonts.

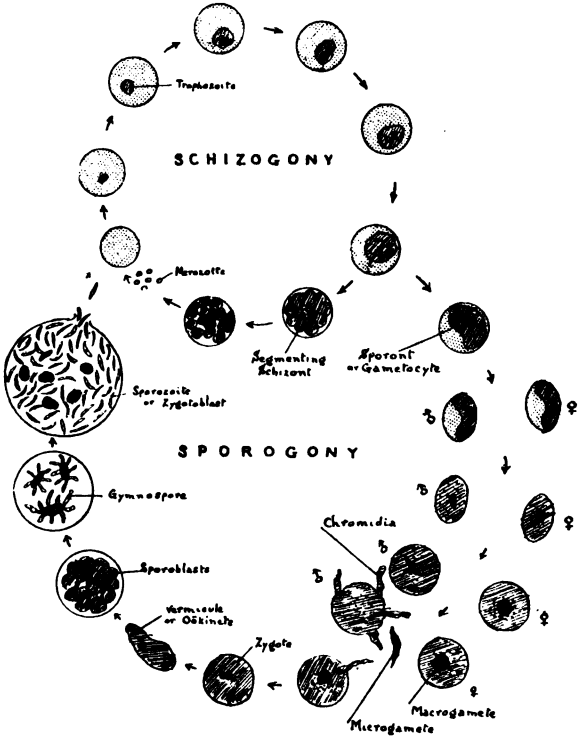

FIG. 12.—THE MALARIAL PARASITE.
Diagram of Endogenous and Exogenous Cycles.

In the male the nucleus is central and the pigment granules scattered. In the female the nucleus is also central, but with pigment aggregated around it. Both male and female are crescent or sausage shaped in the case of the malignant parasite (*H. præcox*); while in the two benign varieties the sexual sporonts are oval or irregularly round.

If not now ingested by a mosquito, the male sporonts die, but the female can lie dormant, and can start a schizogonous cycle by parthenogenesis, thus causing a clinical relapse.

If, however, they are taken up by a mosquito, further development takes place within that host as follows: The nucleus of the male sporonts gives off elements called *chromidia*, which are extruded from the parasite as flagella, and become detached (*microgametes*). These seek the female sporont, now called a *macrogamete*, and, entering it, the nuclei are fused, and the fertilized parasite is known as a *zygote*. The zygote elongates and becomes motile, and is then called a *vermicule* (*oökinete*). The vermicule pierces the stomach of the mosquito and becomes encysted, growing steadily in size until, at about a week or more after being ingested by the mosquito, *sporoblasts* are formed by division of the vermicule nucleus. Further division occurs in the nucleus of each sporoblast, and these subnuclei travel to the surface and project as protoplasmic protrusions, known by the name of *gymnospores*. These gymnospores, when detached, form spindle-shaped sporozoites, which work through the tissues of the mosquito until they reach the salivary glands, and from thence can reach a new human host when the mosquito in question next feels carnivorously inclined.

Thus the exogenous cycle (or sporogony) is completed.

The following table will give the morphological data of the three parasites:

	H. Vivax.	*H. Malariæ.*	*H. Præcox.*
Schizogonous cycle ...	Forty-eight hours	Seventy-two hours	Variable
Red corpuscles	Pale and enlarged	Normal in size and colour	Indefinite
Pigment ...	Extensive, but fine	Coarse	Scanty
Merozoites ...	Twelve to twenty-four	Nine to twelve	Variable and few
Sporulation site	Peripheral blood	Peripheral blood	Liver, spleen, and bone marrow
Scüffner's dots (fine granules in infected corpuscle) ...	Present	Absent	Absent
Sporonts ...	Circular or oval	Circular or oval	Crescent or sausage shaped
Period of latent infection ...	Fourteen days	Twenty-one days	Ten days

Symptoms.—During the latent period of infection the parasites are quickly multiplying by schizogony, and when a certain stage is reached the body reacts and fever results.

The fever is periodic, each access synchronizing with a fresh sporulation of parasites in the blood.

Thus, in the case of benign or malignant tertian, paroxysms will occur at intervals of about forty-eight hours, that being the necessary interval for the completion of each generation of parasites.

If two infections have occurred on two following days, then the type of fever will be quotidian, for a generation will attain maturity in the blood each day. The same quotidian effect is said to be occasionally obtained by the action of quinine on a single malignant tertian infection.

In the case of simple quartan infections the pyrexial attacks will occur at intervals of seventy-two hours, thus leaving two days without fever between each exacerbation.

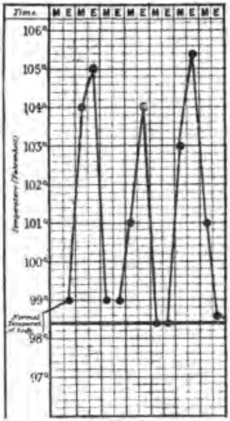

FIG. 13.—BENIGN TERTIAN.

Apart from the periodicity of the fever, the clinical symptoms of the two *benign infections*—tertian and quartan—are much the same.

The onset is somewhat sudden. A headache may precede an early rigor, and rapidly becomes worse.

Nausea and vomiting are not uncommon. The pulse is small and fast, and of fairly high tension.

After half an hour to one hour, with the temperature rapidly rising, the rigor passes, and subjective sensations of heat are felt, and are known as the 'hot stage.'

The temperature now reaches its maximum of 104° or 105° F., and after continuing for four or five hours, a crisis occurs with profuse diaphoresis, and all the sypmtoms are abated.

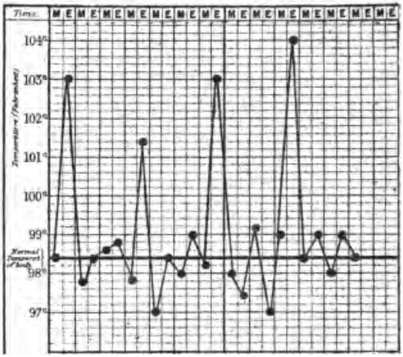

FIG. 14.—QUARTAN FEVER.

The beginning of the fever and the rigor synchronize with the segmentation of the parasite (rosette bodies), and the maximum temperature and hot stage with the release of the merozoites.

If untreated, the paroxysms occur with regularity.

In the case of *malignant infection* the symptoms are more aggravated. The fever is rather remittent than intermittent. Delirium and typhoidal states are frequently met with. Relapses are common, and serious cachexia is usually established.

Two other types are often seen in these pernicious attacks:

A *gastro-intestinal type*, in which there may be intense gastric irritability, with jaundice, passing often into a dangerous typhoidal condition ; or the lesions may occur lower in the alimentary tract, and a dysenteric or choleraic condition be set up.

The second abnormal type is the *cerebral*, in which the usual but severe paroxysms, with vomiting and delirium, give way to drowsiness and coma, with a rapid, small, and irregular pulse, absent reflexes, dilated pupils, and sometimes meningitis, aphasia, amblyopia, etc.

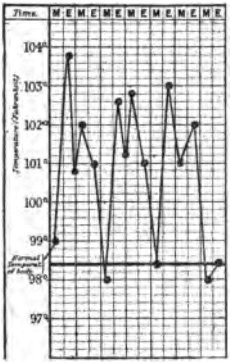

FIG. 15.—MALIGNANT TERTIAN.

In dealing with malaria, the *diagnostic points* are:

1. The presence of the parasite.
2. The presence of pigmented leucocytes.
3. A mononuclear leucocytosis.
4. An enlarged spleen.
5. Response to quinine.

The *prognosis* is favourable in the benign infections, but should be guarded in the malignant type unless treated early.

A condition of *malarial cachexia* is apt to follow repeated attacks. The spleen is chronically enlarged; there is anæmia of the pernicious type; there is much diaphoresis, insomnia, anorexia, emaciation, and weakness; the skin becomes clay-coloured; a chronic parenchymatous nephritis is frequently induced, with œdema and albuminuria; while, in advanced cases, keratitis or optic neuritis is not uncommon.

Treatment.—Quinine is a specific. It is rapidly absorbed, and should be administered about an hour before a rigor is anticipated, in order to kill off the generation of merozoites about to be liberated. In benign infections 5 grains are ample to kill off a generation. In malignant attacks double doses can be given with advantage, and the treatment should always be prompt.

Coma or gastric irritability will contra-indicate its exhibition by the mouth, and the following should be given at once:

℞. Quin. bihydrochlor. ... ... gr. xv.
Aq. destil. ... ... ʒi.
M. Ft. sol. hypoderm.
Sig.: To be injected deeply into scapular or gluteal muscles, and repeated several times if necessary.

In giving quinine by the mouth, it should be remembered that the sulphate is very insoluble. The most soluble form is the bihydrochlorate (1 in 1); after which come the bihydrobromate (1 in 7) and the bisulphate (1 in 11).

When the fever is ended, an iron and arsenic pill can be given with advantage:

℞.	Ferri hypophosph. … … …	gr. ii.
	Quin. bisulph. … … …	gr. i.
	Acid. arseniosi … … …	gr. $\frac{1}{60}$

M. Ft. pil. Sig. : Pil. i. t. d. s. p. c.

In malarial cachexia, arsenic and red marrow tabloids are of more value than iron, since they increase the output of red corpuscles.

Prophylaxis.—

1. Do not live too near natives.
2. Use a mosquito-net.
3. Abolish mosquito breeding-places.
4. Use quinine.

Pathology.—The *anæmia* is threefold:

1. Diminished volume.
2. Diminished hæmoglobin value.
3. Oligocythemia.

There is a mononuclear leucocytosis.

The *spleen* is enlarged, soft and black, the pulp being crowded with infected corpuscles.

There is marked phagocytosis; and amyloid changes may be present in old-standing cases.

The *liver* is often rather enlarged, with a perilobular fibrosis in chronic cases.

The *brain* frequently exhibits punctiform hæmorrhages. The grey matter is darkened. The capillary endothelium is swollen, and the lumen crowded with infected corpuscles, forming the emboli which give rise to the pernicious cerebral symptoms.

The *bone marrow* is pigmented both in the blood-vessels and in the parenchymatous cells.

In rare instances the kidneys or gastro-intestinal tract may be the seat of a localized infection.

Technique of Blood Examination.

A drop of blood is obtained by pricking (after cleansing) the finger or ear lobule of the patient.

This blood may be examined either as a fresh film or a dried and stained film.

Fresh Films.—

Method 1.—Take up the drop of blood on the centre of a coverslip, and drop it at once on to a slide.

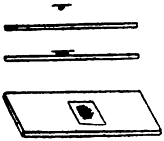

FIG. 16.

Method 2.—Cement a coverglass on to a slide with paraffin wax or vaseline, as shown in the illustration, and approximate the edge to the drop of blood, which will spread by capillary attraction.

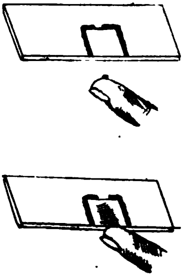

FIG. 17.

Dried Films.—This is a useful way, since the films can be stored or transmitted for subsequent examination or staining.

The best method is to take up the drop of blood on the under edge of one end of a slide. The drop should then be brought into contact with another slide near its end, and, after the drop has properly spread by capillary attraction, the first slide should gently be pushed along the surface of the second, holding it meanwhile at an angle as shown in the annexed cut. An acute angle will give a thin film; an obtuse, a thick one.

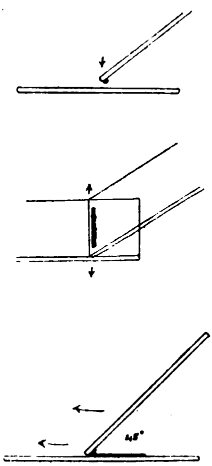

FIG. 18.

After being dried, the films must be fixed by immersion for five minutes in equal parts of alcohol and ether.

They can then be stained; *eosin* and *hæmatoxylin* give excellent results.

1 Stain for half a minute in a 0·5 per cent. alcoholic solution of eosin.
2. Wash, and while wet—
3. Stain in filtered hæmatoxylin for half a minute to ten minutes.

Hæmatoxylin	1 gr.
Alum	10 grs.
Alcohol...	5 c.c.
Dist. water	100 c.c.
Camphor	Small piece.

M.

Use after two months. Keeps well.

Dilute with equal volume of water for use.

Probably the best stain of all for blood films is *Leishman's stain.* It can be procured from Grubler or Burroughs and Wellcome.

0·15 grm. of the dried stain is dissolved in 100 c.c. of pure methyl alcohol.

The films require no preliminary fixing.

A few drops of the stain are put on the film for one minute.

Double the quantity of distilled water is then added and allowed to remain on the slide for five minutes, after which it is washed off, and the film left in pure distilled water for one minute before being dried and mounted. In successful Leishman films the appearances are:

Red corpuscles—pink.
Leucocyte nuclei—red.
Bacilli—blue.
Malarial parasites—blue, with ruby-red chromatin.
Trypanosomes — purple, with red nucleus, centrosome and flagellum.

XXVI

MALTA FEVER

Definition.—A specific infectious fever characterized by an extended duration, frequent relapses, copious diaphoresis, and frequent rheumatic manifestations.

Synonyms.—Rock fever; Gibraltar fever; Mediterranean fever; gastric remittent fever; undulant fever.

Geographical Distribution.—First definitely described by *Marston* in 1859.

It is very common in the Mediterranean islands and on the littoral.

It is also found in India, China, the West Indies, and both the American continents.

Etiology.—The disease is due to invasion by the *Micrococcus melitensis*, first discovered by *Bruce* in 1887.

The following is its *morphology:*

It is a coccus about 0·33 μ in diameter.
Usually occurs singly or in pairs.
Is non-motile.
Is decolorized by Gram.
Flagella have been described.
Grows best at blood temperature on slightly alkaline nutrient media.
Does not liquefy gelatin.
Can live for long in dry state.

Fulfils all Koch's postulates.
Is agglutinated by specific serum in dilutions of 1 in 1,000.
Inoculation gives same disease in monkeys.

The disease is most frequently contracted by new arrivals, and is especially common during youth and middle age.

One attack confers considerable immunity.

The *method of infection* is somewhat uncertain. As the coccus is only found in small numbers in the peripheral circulation, it is unlikely that mosquitoes or biting flies act as intermediate hosts.

The disease is non-contagious. Dust contaminated with infected urine failed to infect monkeys, so that a dust-borne origin is improbable.

Goats are highly susceptible to the disease, and many are found to be infected in the endemic areas. Their milk contains the organism, and thus the ingestion of goat's milk is the most likely solution of ordinary infection.

The urine is the principal vehicle for the escape of the organism from the body.

Symptoms.—There is an *incubation period* of five to fifteen days; and some *prodromata*, such as headache, insomnia, anorexia, etc., are usually present.

There are three chief clinical types of the disease. The *malignant* type is not very common, and is characterized by fever of a high continued type, such as that of typhoid, with pulmonary congestion or basal pneumonia. It is very acute, and may cause death from hyperpyrexia in five to twenty-one days.

There is also an *intermittent* type, with insidious onset and mild course and no initial rigor. This may continue for months, accompanied by night sweats, constipation, anæmia, and debility.

The most common type, however, is the ***undulatory***, of which the following is the usual clinical course:

An initial rigor, with severe headache, facial congestion, and sometimes epistaxis, usher in the disease.

The temperature rises gradually for three or four days, being higher in the evening than morning.

There is epigastric oppression, constipation, and a coated and flabby tongue, with red tip and edges.

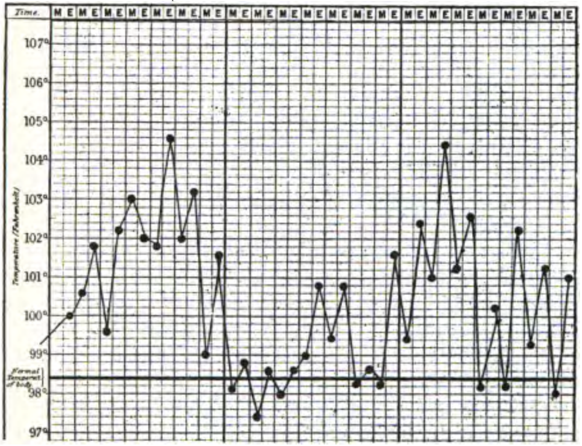

FIG. 19.—MALTA FEVER WITH FIRST RELAPSE.

The liver and spleen, especially the latter, are enlarged.

Each night a profuse diaphoresis occurs, with development of sudamina, but no rose-red spots or tympanites as in typhoid.

After a week or two the symptoms abate, the temperature becomes normal, and the patient feels convalescent. But the night sweats and emaciation continue, and in a few days the first of a long series of relapses begins. During these relapses rheumatoid troubles are very commonly met with. Usually one joint is affected, most often the shoulder, hip, or knee. There is swelling, effusion, and excruciating pain, but no redness. This disappears in three or four days, but may appear in another joint.

Orchitis or epididimitis occurs as a late symptom in about 5 per cent. of the cases.

Albuminuria is rare.

These waves of fever follow each other, extending the disease up to about 300 days, although the average is between 60 and 70 days. Much emaciation, anæmia, and debility naturally ensue, and only improve during the final convalescence.

There is considerable bacillary toxæmia during the later stages, causing a cardiac condition similar to that in diphtheria.

The *prognosis* is usually favourable, the *case mortality* being only 2 or 3 per cent.

Treatment.—This is empirical. A purge should be administered at the outset.

Milk diet, rest, and great care in nursing will be necessary.

Temperature should be kept within bounds by sponging or cold-pack.

Of drugs, quinine is useless. The joint pains are unaffected by salicylates, and therefore hypodermics of morphia will be necessary. If there is much sleeplessness, chloral, bromides, or veronal, will be indicated. Cardiac weakness will need stimulants or digitalis.

Great care and precautions against chill should be taken during convalescence, when tonics and change of climate will be indicated.

Pathology.—The *spleen* is enlarged, hyperæmic, and soft, with enlarged Malpighian bodies and much specific bacterial invasion.

The *liver* is enlarged and congested, with round-celled infiltration of the interlobular tissue. The *M. melitensis* is found therein in large numbers.

The *large and small intestines* show patchy congestion, but the solitary glands and Peyer's patches are normal.

The *lungs* usually show hypostatic congestion, and may be hepatized.

XXVII

ORIENTAL SORE

Definition.—A tropical specific granuloma of the skin characterized by indolent ulceration.

Synonyms.—Delhi boil; Sahara chancre.

Geographical Distribution.—The disease occurs throughout Northern Africa, Mediterranean islands, Southern Russia, Syria, Arabia, Persia, Northern India, and Brazil.

It is not found everywhere in the above regions, but affects certain towns and districts.

Etiology.—In 1885 *Cunningham* found some minute bodies in the exudate of the lesion, and these were demonstrated by *Wright* in 1904 (by the more advanced methods of modern staining) to be identical with the Leishman-Donovan bodies which are the specific cause of kala-azar.

The majority of lesions occur on the uncovered parts of the body. They are very chronic, and may last for a year.

The disease is contagious and auto-inoculable.

The attack confers immunity. The intermediate host, if any, has not yet been discovered.

Unlike kala-azar, the disease is local, and is not followed by constitutional disturbance.

Symptoms.—After an incubation period of three to twelve days, a primary itching papule appears, usually on the legs or arms. After a few days this papule becomes covered with a scab which, when removed, discloses a shallow ulcer. The ulcer is congested at the periphery, has an irregular surface with a jagged edge, and discharges a thin fluid. It gradually increases in size, and is accompanied by troublesome itching, but seldom by pain.

One or two such ulcers are the rule, but twenty or more are sometimes seen on one person.

Cicatrices form after some months, and are depressed and often pigmented.

Constitutional disturbance is absent, and the prognosis favourable.

Treatment.—Early excision of the papule is indicated; also tonics for anæmia.

Pathology. — The condition is a chronic serofibrinous inflammation, with a round-celled infiltration of the skin and subcutaneous tissue. In the centre of this necrosis proceeds; the hair root-sheaths are cornified and the bloodvessels obliterated.

Leishman-Donovan bodies are found in the exudate at the base of the lesion.

XXVIII

PELLAGRA

Definition.—A disease of limited distribution, characterized by an erythematous exanthem.

Synonyms.—Mal de sol; lepra italica.

Distribution.—It is endemic in Spain, Italy, South-West France, Egypt, Algiers, and Mexico.

History.—It was first reported by *Casal,* in Spain, in 1735.

Etiology.—Middle age is the most frequent time for attack, and it is chiefly a malady of the lower classes.

The disease would seem to be an intoxication, and is thought to be due to the eating of damaged maize, but the evidence on this point is inconclusive.

Sombroso considers a chemical change to occur in the maize, as the result of the growth of a non-pathogenic micro-organism.

The reasons in favour of the theory are:

1. That maize is the staple food in pellagra countries.
2. That extracts from damaged maize will cause similar symptoms in animals and man.
3. That the disease only occurred after the introduction of maize into Europe.

The latter fact is, without doubt, incorrect, as maize was certainly eaten in Italy 200 years before the advent of pellagra.

It is probable, however, that insanitary surroundings and unwholesome food are predisposing causes.

Symptoms.—Languor and debility are frequent prodromata. After some months dyspepsia and diarrhœa may commence, frequently accompanied by headache, vertigo, erotic perversions, etc.

This is followed by an erythema of face, neck, chest, hands, and dorsum of forearms.

Improvements, followed by exacerbations, continue, until in the course of a few years cerebro-spinal symptoms develop, with paralysis, spastic gait, melancholia, etc.

Cachexia results, with marasmus, bedsores, etc., which eventually kills the patient.

The duration of the disease may extend to fifteen years ; the prognosis is gloomy.

Treatment.—Arsenic internally. Diet, nutritious and liberal.

Pathology.—Fatty degeneration of liver. Spleen atrophic. Kidneys cirrhotic. Sclerosis of posterior and postero-lateral cord. Meningeal inflammation frequent. Peripheral nerves normal.

XXIX

PEMPHIGUS CONTAGIOSUS

Definition.—A tropical eruption of a vesicular type, the bullæ of which are transmitted by inoculation through the medium of hands or clothes.

Distribution.—It is reported from certain parts of India, Malaya, and China.

Etiology.—The disease is thought to be allied to *pemphigus neonatorum*, and is probably due to a pyogenic streptococcus, which would account for the facility with which auto-inoculation occurs.

Recently Leishman-Donovan bodies are reported to have been found in the bleb contents.

The disease may occur at any age, but is probably commonest in children.

Symptoms.—A hemispherical vesicle first appears, and soon becomes flaccid, with cloudy contents.

From this source of infection the disease may be spread widely by clothes or hands.

The blebs heal spontaneously, and new epidermis is found underneath.

Treatment. — Limited to antiseptic lotions and powders.

XXX

PHŒBISM

Definition.—Phœbism is a condition akin to shock, caused by the actinic rays of the solar spectrum, and characterized by meningeal congestion and cerebral or cerebro-spinal disturbances.

Synonyms.—Sunstroke; insolation; sun traumatism.

Geographical Distribution.—It may occur anywhere, but, naturally, is especially a matter of tropical distribution.

Etiology.—The condition is one of cerebral shock, caused by exposure of the head or spine to the ultra-violet rays of the spectrum, in an unacclimatized person.

It is an acute cerebro-spinal congestion, sometimes followed by chronic meningeal inflammation.

Acclimatization may be gradually acquired, and seems to be hereditarily so amongst natives.

New-comers and Europeans are, therefore, more likely to be attacked.

Unsuitable headgear and clothing will strongly predispose to the condition, as also alcoholic indulgence.

Symptoms.—Slight cases will be familiar to all.

Undue exposure to a tropical sun, causing an extensive sunburn, often some painful exudation, or even considerable œdema, headache, and slight febrile reaction.

The more severe cases are not uncommonly seen amongst soldiers or sportsmen. There is a more or less sudden loss of consciousness, with a moist skin and small, low-tension pulse.

The temperature is normal or subnormal, and the breathing is heavy, or even stertorous.

Such an attack will end in one of the following ways: Either recovery takes place, with no injurious effects; or an acute meningitis may supervene; or gradually a chronic meningeal inflammation may be brought about, leading to various cerebral disturbances, such as amnesia, deafness, paresis, epilepsy, or dementia.

In certain cases the shock to the nerve centres may be so great that sudden death from heart failure may occur.

Treatment.—The patient should be removed to a cool, dark room; ice applied to the head, and hot bottles to the feet; a croton-oil purge is advisable.

The condition of the heart may indicate the necessity for the exhibition of digitalis or alcohol.

When consciousness is recovered, bromides should be administered.

During convalescence, light and digestible food will be necessary, and a change to a cool climate will be essential if the condition has been a grave one.

In the more serious cases no return to the tropics should be allowed.

Pathology.—In cases of sudden death, cerebral hyperæmia will alone be found.

In post-mortem examinations on chronic cases the diploë are found to be obliterated; the meninges are thickened, tough, and adherent; the convolutions are flattened, and there is sometimes some cerebral softening.

XXXI

PINTA

Definition.—A tropical skin disease of trichophyton origin, characterized by piebald discolorations of the skin.

Geographical Distribution.—First reported in Mexico in 1775, and is endemic in that country, as also in Central America and several South American republics, the chief seat being in Columbia.

Isolated cases have been noted in the Gold Coast, Tripoli, and Egypt.

Etiology.—The specific cause is the *Trichophyton pictor*, a fungus with a tapering branched mycelium having conidia attached.

The spores are roundish, and about 10 μ in diameter.

The disease does not seem to be directly contagious, and may possibly be conveyed by mosquitoes or bugs.

There are three differential forms of the fungus—violet, red, and white. The former variety attacks the epidermis, and is common amongst the negroes in rural districts; there is a late loss of pigment.

The red variety affects the rete and corium, is common amongst white people in towns, and is followed by no loss of pigment.

The white kind has a like skin affinity to the red; the town half-castes are the chief sufferers.

Symptoms.—The incubation period is apparently about one month.

The eruption begins on the face or neck, with asymmetrical patches or spots and furfuraceous desquamation.

The patches increase and coalesce, showing the characteristic colours after two or three years.

The white variety should not be confused with Vitiligo (*q.v.*).

The mucous membranes may become coloured during the chromatic stage.

The disease, though it may last for very many years, is benign; and there are no constitutional symptoms.

One or more colours are sometimes seen on the same patient.

Treatment.—

℞. Chrysarobini ... . ʒi.
Ol. caryophylli ... ... ♏ x.
Ung. paraffin. ... ... ℥i.
M. Ft. ung.

XXXII

PITYRIASIS NIGRA

Definition.—A tropical skin disease of microsporon origin.

Distribution.—Chiefly found in Ceylon, Malaya, and Southern China.

Etiology.—The specific cause is the *Microsporon mansoni* (Castellani). The mycelial threads are non-branching, short, and irregular in outline, 20 μ long by 2½ μ broad.

The spores are globular, large (5-7 μ), and arranged in clusters (Castellani).

Symptoms. — Manifested by round, dull-black patches, which desquamate and do not itch.

It may occur on any part of the body, but appears to avoid the face. It has a partiality for the neck and region of the axilla.

Treatment.—

℞. Ichthyol Sulphur	... ... āā 5 parts.
Vernisol...	... ... 90 parts.

M. (Keep well corked.)

Sig.: Smear a small quantity on the desired area and allow to dry.

XXXIII

PLAGUE

Definition.—An acute specific infectious disease, characterized by fever, severe constitutional disturbance, and either a specific adenitis, pneumonia, or septicæmia.

Synonyms.—Black-death; *pestis bubonica.*

Geographical Distribution.—Southern Thibet was probably the original centre.

Great pandemics have originated on many occasions in India or China, and spread by trade routes over wide areas of the globe.

One such visitation swept almost the whole globe in the fourteenth century, being known as the Great Black Death.

In the seventeenth century Europe was again visited in serious epidemic form for the last time, and is known in English history as the Great Plague of London.

The present pandemic began in the province of Yunnan, in China, in about 1890. It spread gradually through China and via Hong-Kong to India. Bombay was reached in 1896, and Calcutta in 1897. It then visited Mauritius, Madagascar, East Africa, Suez, Alexandria, Japan, the Philippines, Australia, Paraguay, Brazil, and the Argentines. In India alone during the year 1904 there were over 1,000,000 deaths from this cause.

Etiology. — The disease is caused by a micro-organism, the *Bacillus pestis*, discovered in Hong-Kong by *Kitasato*, in 1894, and soon after by *Yersin*, independently.

The following are the *morphological characteristics* of the microbe:

A round-ended rod, almost coccoid.
1·5 to 2 μ in length by 0·7 μ in breadth.
Non-motile.
Has one or two terminal flagella.
Does not form spores.
Is a facultative anaërobe.
Is decolorized by Gram.
Does not liquefy gelatin.
Coagulates milk in two weeks.
Exhibits bipolar staining.
Haffkine's diagnostic culture, viz.:—

> If cultivated in a faintly alkaline peptone broth, covered with clarified olive-oil, and kept at rest, in a day or so colonies will form a stalactite growth depending from the under surface of the fat.

Is very resistant to cold, and can be frozen for some months without injury.
Is killed by sunlight in one hour, by heat of 80° C. in five minutes, and fairly easily by most disinfectants.

Infection may occur in three ways:

1. *By the respiratory tract.*
   It is by this means that the less common type of pneumonic plague (in which the lungs are the primary focus of infection) is conveyed from person to person.
2. *By the gastro-intestinal tract.*
   The less common primary septicæmic cases will be thus conveyed.

3. *By the broken skin or insect-bites.*
   The commonest type of plague is the bubonic form, and this form is generally conveyed by flea-bites, or the inoculation of abrasions by infected material.

The species and habits both of fleas and rats are important in this connexion.

Fleas.—Certain species of fleas show predilection for different hosts. If their host dies or is ill, they seek another of the same species. If such is not available, the nearest warm-blooded animal will be made use of.

Epizoötic Host.	Species of Flea.	Biting Habits.
Man	*Pulex irritans* ...	Chiefly nocturnal
Cats Dogs Monkeys Sheep, etc. ...	*Ctenocephalus canis and C. felis* ...	Largely diurnal
Black rat (*Mus rattus*) ...	*Loemopsylla cheopis*	Chiefly nocturnal
Large brown rat (*Mus decumanus*)	*Ceratophyllus fasciatus*	Chiefly nocturnal

Rats.—There are many species of rats, and all are highly susceptible to plague. Human epidemics of plague are frequently to be traced to a preliminary epidemic amongst the local rats. To understand this, the habits of rats should be appreciated, as well as those of fleas.

The black rat (*M. rattus*)—common in India and many other places—is a house rat, and lives much in close proximity to man. This rat was very common in Europe up to the end of the seventeenth century, when the large brown rat invaded the Continent and quickly displaced the black rat.

The brown rat (*M. decumanus*) lives especially in roofs, cellars, and drains, and is by no means the close neighbour of man that the black rat is.

When rats find any numbers of their colony to be ill or dying, they migrate in a body to some other haunt, leaving the sick and dead behind.

The fleas from these deserted rats seek a fresh host. The black rat being a near neighbour of man, the fleas from such rats will seek man, and thus start a human epidemic. If, on the other hand, the rat epidemic had been amongst brown rats, then the chances of human infection by their fleas is naturally enormously minimized.

The disappearance of pandemic plague from Europe is thus reasonably explained, as also the comparative immunity of Singapore and other such places in which the black rat is a *rara avis*.

With regard to the *incidence* of the disease, neither race, sex, nor age are influencing factors.

Personal and domestic cleanliness and freedom of house and person from vermin will naturally explain the small case incidence amongst Europeans.

Symptoms.—There is an *incubation period* of one to three days, but in dealing with large bodies of natives, such as immigrants, troops, etc., a quarantine period of at least ten days should be insisted on, unless a remarkably thorough disinfection of clothes and effects and persons has been undertaken before leaving an infected area.

Prodromata are not common. The onset of the disease is sudden, and may be exhibited as convulsions in the case of children.

The temperature quickly rises. The face and conjunctivæ become flushed and injected.

The expression is extremely anxious, and the gait reeling and drunken.

The tongue is swollen and furred, with red tip and edges, and there is intense frontal headache.

The pulse is fast and full, but of unexpectedly low tension.

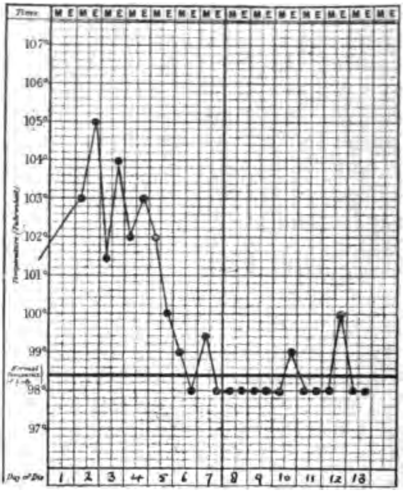

FIG. 20.—BUBONIC PLAGUE: RECOVERY.

Very soon a gland, usually one of the groin glands, becomes swollen, indurated, and painful.

The temperature usually reaches its maximum on the third day. The pulse, which is still fast, becomes dichrotic.

A lethargic apathy supervenes, with thick speech or slight delirium, scanty urine, and dusky face.

This is a critical period of the disease. The temperature and pulse-rate may diminish steadily in favourable cases until normal is reached in seven or eight days.

If the temperature does not fall below 100° F., and tends to rise, as also the pulse-rate, it is an indication of probable invasion of the blood, and fatal septicæmic features will supervene.

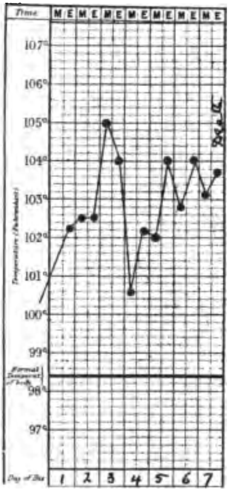

FIG. 21.—BUBONIC PLAGUE: SECONDARY SEPTICÆMIA.

Or, again, after the maximum temperature has been reached, there may be a sudden subnormal fall, with collapse and death.

In the bubonic and pneumonic types of the disease the bacillus is not found in the peripheral blood, except just before death in fatal cases. In the septicæmic cases it will be found throughout.

There is usually a marked leucocytosis, of polynuclears, the lymphocytes being also increased.

In the *pneumonic type* the initial features are the same as those of the usual bubonic disease described above; instead, however, of a bubo making its appearance, the respiration becomes accelerated and is accompanied by a cough. The sputum is not rusty, as in lobar pneumonia, but may be blood-streaked, and is loaded with bacilli in nearly pure culture. There will be dullness on percussion, and rhonchi will be heard on auscultation. It is a very fatal type of disease, and death takes place on the third or fourth day.

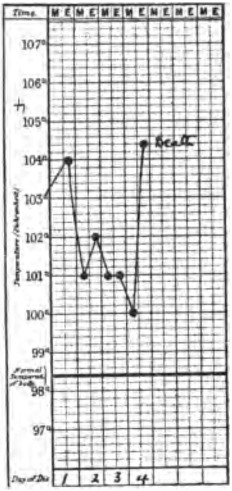

FIG. 22.—PNEUMONIC PLAGUE.

In addition to the three clinical types mentioned above, there is a fourth mild or ambulatory type, known as *Pestis minor*.

It is characterized by a slightly enlarged groin gland, rather tender, but not indurated. There is a minimum of constitutional disturbance; in fact, patients may move about as usual.

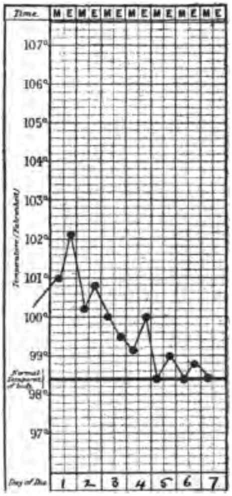

FIG. 23.—PESTIS MINOR.

The temperature rises, but is seldom above 101° F., and rarely lasts more than three days.

These cases may precede an epidemic, or may follow one as an indication of diminished virulence. They are naturally a grave source of danger to the community, since they so easily pass unrecognized, and yet are capable of distributing the disease.

The *case mortality* will vary in different epidemics.

In the usual bubonic type the mortality is, as a rule, about 70 per cent. amongst natives, and 20 per cent. amongst Europeans.

The septicæmic and pneumonic types are almost always fatal.

Amongst pestis minor cases the mortality will be practically nil.

The *complications and sequelæ* may be varied.

Pregnant women always abort.

There is often extensive sloughing at the site of the bubo, sometimes extending to the bone.

Convalescence is always protracted, and the subsequent prostration is extreme.

Treatment.—There is no specific drug. There is a Hong-Kong treatment which seems to be efficacious in reducing the mortality—viz., large doses of pure carbolic acid.

Fifteen grains are given every two hours. Carbolic poisoning is rare. If carboluria supervenes, it can be remedied by the omission of a few doses.

Symptoms should be treated as they arise.

There are five chief *therapeutic sera*—those of *Yersin*, *Lustig*, *Terni*, *Bondi*, and *Kitasato.*

The first two are those chiefly in use.

Yersin's serum is obtained from a horse immunized by repeated injection of living cultures. To be of any therapeutic avail, it must be used during the four days of the disease.

Lustig's Serum.—A nucleo-proteid, soluble in a solution of sodium carbonate, is obtained by dissolving an agar cultivation in caustic potash and acidifying with hydrochloric acid.

Of the *prophylactic sera*, Haffkine's is the most generally employed.

A broth cultivation of the organism is sterilized by heat, and carbolic acid is added.

An injection will cause a slight rise of temperature, with some headache and malaise, perhaps lasting for a day or two.

By this means protection for seven or nine months is secured.

The Indian Plague Commission found that in the incubation stage it frequently aborted the disease; in the healthy a strong protection is afforded; and amongst those attacked, a large percentage of the inoculated recover.

Pathology.—The bubo appears dark red on section, with the tissues frequently pulpy and soft. There is periglandular infiltration and hæmorrhage.

Microscopically the tissues are infiltrated with leucocytes, red corpuscles and bacilli.

Other glands in the same chain are usually implicated.

The *spleen* is congested, soft, and enlarged, and microscopically is loaded with bacilli.

The *kidneys* are congested and enlarged, but do not contain many bacilli as a rule.

The *liver* is very congested, and sometimes enlarged, and in septicæmic cases exhibits necrotic foci.

The *lungs* are congested and œdematous in bubonic plague; while in the pneumonic variety there is a confluent lobular pneumonia.

XXXIV

PRICKLY HEAT

Definition.—An inflammatory skin affection caused by obstruction to sweat secretion.

Synonyms.—*Lichen tropicus; miliaria papulosa.*

Distribution.—General throughout the tropics.

Etiology.—The condition is due to obstruction to the sweat secretion while the body is exposed to increased atmospheric temperature.

It is predisposed to, or aggravated by, the use of flannel garments, iced drinks, and violent exercise.

Symptoms.—It is manifested by an eruption of small, red, acuminate papules, closely approximated, and occasionally interspersed with little vesicles.

The onset is sudden, and the area involved may be large or small.

There is intolerable pricking and tingling.

Scratching may lead to dermatitis or boils.

It generally ceases spontaneously in cool weather.

Treatment.—Dilute with water some glycerine of borax to which thymol has been added, and apply frequently.

Flannel garments should not be worn next to the skin.

Violent exercise and indulgence in iced drinks should be avoided.

XXXV

SEVEN-DAY FEVER

Definition.—A non-malarial sporadic fever of the tropics, characterized by a saddle-back temperature of about a week's duration, a relatively slow pulse, frontal headache and pains in the back, and due probably to a bacillus of the *B. coli* group.

Geographical Distribution.—The disease is a common one in Indian ports, especially Calcutta, Bombay, Madras, and Rangoon, and will probably be found to be a widespread disease in many ports of Egypt, the East, and the Far East.

It does not seem to occur to any extent far inland.

Etiology.—The disease is very probably due to a bacillus cultivated from the blood (by *Rogers*), although not yet definitely proved.

The following is the *morphology:*

It is allied to the *B. coli* group.
Is from 3 to 5 μ long.
Is actively mobile.
Has a few flagella.
Does not liquefy gelatin.
Is decolorized by Gram.
No gas formation in glucose agar.
Invisible growth on potato.

In litmus milk no clotting, and only slight acidity after a few days.

Agglutination reaction (not always constant) occurs, with dilutions of 1 in 20 and 1 in 40.

There are fairly constant *blood changes* in the disease.

The red count is normal, but the leucocytes show a considerable relative reduction.

The polymorphonuclears are relatively and actually reduced.

The lymphocytes and large mononuclears are actually about normal, but are relatively increased.

The *race and sex incidence* is somewhat marked. It is much more common amongst Europeans than amongst natives.

The early cases each year occur chiefly amongst shipping folk, later cases amongst other inhabitants—chiefly new-comers.

It is most common amongst males.

It occurs chiefly between the months of May and September each year.

Symptoms.—There is (like dengue, but unlike typhoid) usually a sudden onset, with rigors in 60 per cent. of the cases.

The face is flushed and the expression dull and listless. Pain in the back is a frequent symptom, also frontal headache.

The tongue is furred dorsally, with raw red edges.

Unlike malignant malaria, there is usually no gastric disturbance.

The abdomen is often distended, and sometimes painful.

The spleen and liver are rarely enlarged, and skin rashes are seldom seen.

The *temperature* rises fairly rapidly, and shows a characteristic 'saddle-back' curve, with two elevations and a termination by lysis, ending at about the end of a week.

Occasionally the type may be a high remittent one resembling typhoid, or the terminal rise may be prolonged and high.

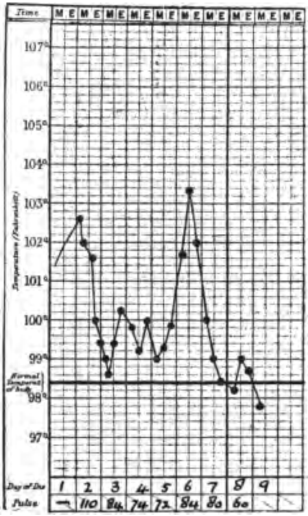

FIG. 24.—SEVEN-DAY FEVER.

(Note saddle-back temperature and slow pulse.)

An important and constant feature of the disease is seen in the *pulse*. This is remarkably slow in comparison with other types of fever. Notwithstanding the pyrexia, it rarely rises above 100 or 110, except during the first few hours of the disease or during a hyperpyrexia.

Convalescence is rapid. *Relapses* are rare, but one attack does not confer immunity.

The *mortality* is practically nil.

Treatment.—Quinine is useless. Aspirin might be useful in controlling the pains. In hyperpyrexia, cold sponging should be employed.

The usual milk diet and other fever treatment is of course necessary.

The **Pathology** has not been worked out.

XXXVI

SPIRILLAR FEVER

Definition.—An acute febrile disease of a relapsing type, due to the presence of a blood spirillum.

Synonyms.—Relapsing fever; tick fever; famine fever.

Geographical Distribution.—The disease is endemic in Bombay, North-West Punjab, Uganda, in the Congo districts, and in Lower Egypt.

Epidemics have occurred in Europe at different times from 1818 onwards.

Cases have been reported from other parts of West Africa, Palestine, and Southern China.

Etiology.—The disease is due to infection by a blood spirochete.

The biological status of these parasites is doubtful.

They are usually considered to be vegetable micro-organisms belonging to the genus Spirillum.

Researches by *Schaudinn* seem to point to a protozoal origin, since, in their extra-corporeal development, they pass through a stage somewhat resembling a trypanosome phase.

Koch shows, however, that they more resemble the Bacteriaceæ in characteristics; that they have no nucleus or undulating membrane; and that division is transverse, and not longitudinal.

As in the case of malaria, more than one species of parasite can give rise to the clinical entity. As far as is known at present, there are only two species incriminated—viz., *S. obermeieri* and *S. duttoni.*

The former was discovered by Obermeier, assistant to Virchow, in 1873; the latter by Dutton and Todd, in 1904.

The following are the differential characteristics:

	S. obermeieri.	*S. duttoni.*
Geographical distribution ...	Family widespread Endemic in Bombay and Lower Egypt	Endemic in Uganda, Congo basin, and probably other regions
Intermediate hosts	Possibly fleas, bugs, lice, or mosquitoes	A tick (*Ornithodorus moubata*)
Susceptibility of lower animals	Benign infection of monkeys, rabbits, rats, and mice	Malignant infection of monkeys, rabbits, rats, and mice, also of dog and horse
Morphological characteristics	Length 16 to 40 μ Two or three spirals One long terminal flagellum Immunizing serum does not protect against *S. duttoni*	Length 13 to 40 μ Two or three spirals Flagella diffuse Immunizing serum does not protect against *S. obermeieri*

With regard to the *incidence* of the disease, it may attack any race, sex, or age. It most frequently occurs between the ages of fifteen and thirty-five. Any insanitary surroundings likely to ingratiate personal vermin will naturally act as predisposing causes.

One attack does not confer immunity.

Staining Technique.—The spirochetes can be stained with the usual dyes, and are unstained by Gram.

For detailed examination Schaudinn's 'syphilis stain' should be used:

Azur II. eosin ...	3·0 grammes.
Azur II.	0·8 gramme.
Glycerine	250·0 c.c.
Methyl alcohol ...	250·0 c.c.

The smear should be fixed for one minute in the vapour of osmic acid, and then stained for ten minutes in a fresh 5 per cent. watery solution of the above stock solution. Afterwards wash in distilled water and dry.

Symptoms.—There is an *incubation period* of three to seven days, after which the disease commences suddenly, and prodromata are rare.

The temperature rises quickly to 103° or 104° F., and remains high for three to five days (*S. duttoni*), or five to seven days (*S. obermeieri*). It is accompanied by much aching of the back and bones, and by great prostration and depression.

Vomiting is a common symptom in the early stages. The tongue is dry and brown, with red edges. The bowels are usually constipated, with occasional post-critical diarrhœa.

The spleen is enlarged in more than 50 per cent. of the cases, and slight enlargement of the liver is common.

After the definite period of continued fever there is a sudden crisis, with copious sweating, usually occurring at night. The urgent symptoms are immediately relieved, and the patient feels almost well.

An apyrexial period of about seven days follows, after which (in about 50 per cent. of cases) there is a sudden relapse, with a repetition of the early symptoms and fever.

Second, third, or even fourth, similar relapses may occur, but are rare, and the type of fever will be more intermittent, and the apyrexial intervals more lengthy.

Unusual and serious types will follow a typhoidal or uræmic course. In the former type hepatic enlargement and jaundice are common.

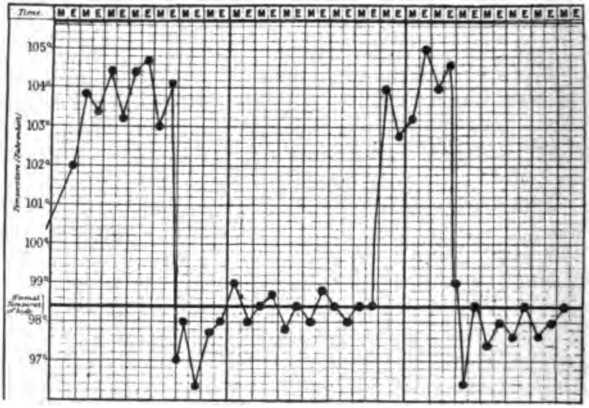

FIG. 25.—SPIRILLAR-FEVER WITH ONE RELAPSE.

The spirilla are only found in the peripheral blood during the period of pyrexia.

The *case mortality* is from 4 to 10 per cent., and in adverse circumstances may rise to 50 per cent.

Treatment.—No drugs have any specific action, and quinine is useless.

Atoxyl might be tried with advantage.

Symptoms should be treated on the usual lines as occasion demands.

Serum-therapy is probably the key to future treatment.

Pathology.—The *spleen* is enlarged, congested, soft, and fatty. Spirilla are present in enormous numbers at all stages of the disease, and are found free, but more often are enclosed in the spleen cells, and cause their degeneration.

The *liver* is enlarged, with cloudy swelling of the hepatic cells, and small-celled infiltration along the branches of the portal vein. Spirilla occur in the cells, and may lead to necrosis.

The *lungs* and *kidneys* are usually congested.

The marrow of the bones is reddened, and the spirillum is found in the marrow cells.

XXXVII

SPRUE

Definition.—A disease of the tropics often becoming chronic, characterized by erosion of the mucous membrane of the alimentary tract, frothy and soft stools, and considerable anæmia.

Synonyms.—Tropical aphthæ; psilosis; Ceylon sore-mouth; Indian hill diarrhœa.

Geographical Distribution. — First reported by *Hillary* from Barbadoes in 1776.

It occurs in other parts of the West Indies, also in Southern China, Malaya, Ceylon, India, and Burma.

Etiology.—The specific agent has not yet been discovered. Several organisms, such as the *Rhabdonema intestinale* and the *Amœba coli*, are frequently found in the stools, but do not seem to be connected pathogenically with the disease.

The condition is found rather more frequently in women than in men, and occurs especially at middle age.

Nearly 90 per cent. of cases are amongst Europeans of long tropical residence.

The disease is never epidemic. Indirect contagion can be traced, however, in some cases.

Symptoms.—There is an insidious onset with anorexia and various dyspeptic manifestations.

An intermittent morning diarrhœa sets in, which later on becomes characteristically copious, pale, and frothy.

Meanwhile raised red spots occur on the edges of the tongue, coalescing and resulting in superficial erosion, which gradually involves the whole buccal mucous membrane. The use of hot food, naturally, is extremely painful, as is also speech.

Emaciation and anæmia follow. The liver becomes atrophied. The urine is diminished in quantity, and loaded with urates.

The attack continues for a year or two, with occasional improvements, and will then terminate in death from intercurrent disease or exhaustion.

On the other hand, it may become chronic and continue for years.

Under proper treatment the prognosis is not unfavourable in recent cases; but if untreated, or in aged people or long-standing cases, the disease is generally fatal.

Treatment.—No drugs are of any specific use. Treatment must be dietetic, and the most widely adopted is that known as the *milk treatment*.

The patient is given a dose of castor-oil and sent to bed. When the bowels have acted, the administration of milk is begun, 3 pints being given daily, by teaspoon, at hourly intervals. When the mouth gets less sore and the stools more solid, the amount of milk should be increased by ½ pint daily until 5 pints per diem are consumed. This amount should be continued for ten days.

The patient is then allowed to get up, and the daily portion of milk is increased to 7 pints, at which amount it is administered for a further six weeks.

After this return to normal diet may be gradually secured.

The use of strawberries is allowed throughout.

Other dietetic treatments are:—(*a*) The *meat treat-*

ment, which consists of the administration of 5 ounces of pounded and lightly cooked beef three times daily, with beaf-tea or jelly every two hours.

(*b*) The *fruit treatment*, in which large quantities of all the less acid fruits form the staple diet.

If drugs are given at all, *chinosol* may be tried in 5-grain doses t. d. s.

Fatigue and chills should be carefully avoided, and tonics are indicated during convalescence.

Pathology.—There is general atrophy and anæmia of most organs.

The characteristic lesions are in the alimentary tract.

There is a primary infiltration of the buccal submucosa.

The bowel is much thinned and the muscular coat atrophied.

There is erosion of the mucous membrane throughout the whole tract. The villi, solitary glands, follicles of Lieberkühn, and Peyer's patches, are all atrophied, and sometimes have almost disappeared.

XXXVIII

TINEA IMBRICATA

Definition.—A tropical skin disease of trichophyton origin, characterized by extensive distribution of tissue-paper-like scales.

Geographical Distribution.—The condition is fairly common in China and the Pacific Islands, and, to a less extent, in Malaya.

Etiology.—The specific cause is the *Trichophyton nansoni*, which grows profusely between the rete and epidermis, causing the latter to peel up.

Morphologically the fungus is much like *T. ectothrix*, but the spores are oval and the mycelium is longer.

It has been cultivated, and can be experimentally transmitted to healthy subjects, with an incubation period of rather over a week.

Symptoms.—A raised, brownish patch appears and spreads peripherally. Other spots succeed the first, and concentric rings are thus formed. Various foci coalesce until a large area is involved, but the crutch, axillæ, and hair usually escape.

Beneath the detached scales the skin is leucodermic.

Pruritus may be annoying.

Treatment.—Commence with a hot bath, and scrub with tar soap. Follow this by—

℞.	Chrysarobini	ʒi.
	Ol. caryophylli	♏x.
	Ung. paraffin.	℥i.

M. Ft. ung.

XXXIX

TRYPANOSOMIASIS

Definition.—A protozoal infection of human beings by a trypanosome (*T. gambiense*), characterized by chronic irregular fever, patchy erythema, anæmia, and enlarged lymphatic glands.

Synonyms.—Sleeping sickness; negro lethargy.

Geographical Distribution.—The chief endemic areas are: Upper Guinea, the hinterland of Sierra Leone, of Liberia, and of the Ivory Coast, French Congo, the Congo Free State, and Uganda.

Cases have occurred in the West Indies, but only as importations from West Africa, and the disease has never spread.

No reports of the disease have been received from other parts of the world.

Etiology.—The condition is a protozoal infection, of which the pathogenic agent is *Trypanosoma gambiense.*

The earliest clinical description of sleeping sickness was given by *Clarke*, of Sierra Leone, in 1840.

The *T. gambiense* was first found in human blood by *Forde*, in 1901, at Bathurst (*Gambia*), and was identified by *Dutton.*

The relation between trypanosomiasis and sleeping sickness was later discovered by *Castellani*, in Uganda, who found the organisms in the cerebro-spinal fluid of a sleeping sickness case.

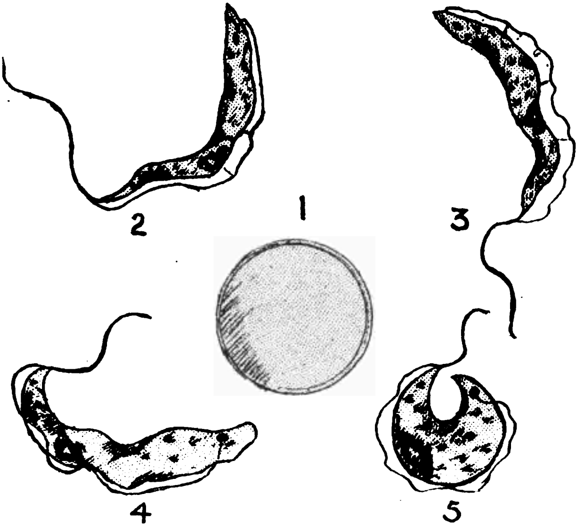

FIG. 26.—T. GAMBIENSE.

The investigation of these protozoa is of great importance as well as interest. The first trypanosome discovered was that of the trout, by *Valentin*, in 1841. Since then the subject has been steadily investigated. At the present time nine species of the genus Trypanoplasma and twenty-nine of the genus Trypanosoma have been described in fishes, of which the *T. remaki* of the pike is the best known, and has a very wide distribution.

Six different species are known to be parasitic in batrachians, and of these the *T. rotatorium* of frogs has a world-wide distribution.

Fifteen species of trypanosomes have been described as occurring in birds, and of these *T. avium* has the most wide-spread distribution.

Of the important mammalian Trypanosomata, the following list will give the chief points of a few pathogenic species which are of economic interest.

In addition to these there are many non-pathogenic species, of which the *T. lewisi* of rats has a world-wide geographical distribution.

PATHOGENIC MAMMALIAN TRYPANOSOMATA.

	Pathogenic Manifestation.	Intermediate Host.	Geographical Distribution.	Morphology.
T. brucei ...	'Ngana,' a fatal cattle disease	A tsetse fly, the *Glossina morsitans*	Southern Central Africa	Length 25 to 30 μ Breadth 1·5 to 2·5 μ Protoplasm has many chromatic granules
T. evansi ...	'Surra,' a cattle disease	Horse-flies (*Tabanidæ*)	India	Length 20 to 30 μ Breadth 1 to 2 μ Flagellum is long
T. equinum ...	'Mal de caderas,' a disease of horses and donkeys	A fly, the *Stomoxys calcitrans*	Central and South America	Length 20 to 25 μ Breadth 2 to 3 μ Centrosome is very small
T. equiperdum	'Dourine,' a disease of horses	Nil (infection by coitus)	Algeria and India	Length 18 to 26 μ Breadth 2 to 2·5 μ No protoplasmic granules
T. theileri ...	'Galziekte,' a cattle disease	A fly, the *Hippobosca rufipes*	South Africa	Length 30 to 65 μ Breadth 2 to 4 μ Distinguished by large size
T. gambiense ...	Human trypanosomiasis; also pathogenic to a very large number of mammals	A tsetse fly, the *Glossina palpalis*	Portions of Equatorial Africa	Length 18 to 25 μ Breadth 2 to 2·8 μ

The method of *human infection* is by means of a biting tsetse fly, the *Glossina palpalis*, which acts as an intermediate host. The spread of the disease will, therefore, depend on the presence of infected patients and the distribution of the proper species of tsetse fly.

With regard to the *incidence* of trypanosomiasis, sex and age have no determining influence. The disease is very largely prevalent amongst poorer-class negroes, but no race is exempt, and seven or eight cases have been recorded amongst Europeans.

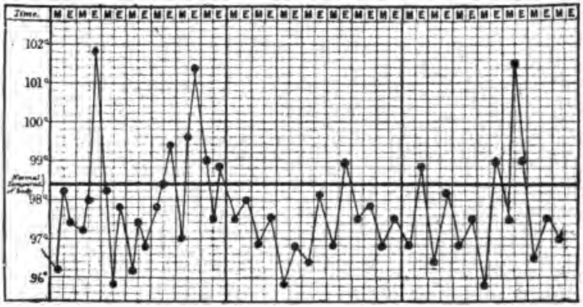

FIG. 27.—HUMAN TRYPANOSOMIASIS. IRREGULAR FEVER OF FIRST STAGE.

Symptoms.—The *latent period of infection* varies from four weeks to a year or more (? seven years).

The actual onset is insidious. There is an irregular intermittent fever (*vide* chart), without rigors or pronounced sweating. This is accompanied by cardiac excitability, progressive muscular weakness, frequent localized œdemas or erythemas, and, very commonly, by enlargement of some of the lymphatics—usually the cervical.

On percussion, the splenic dullness will be found to be increased.

Amongst negro patients the above-mentioned early symptoms are frequently absent, or are not well marked, with the exception of the cervical gland enlargement, which is practically constant.

During this first stage the trypanosomes have not invaded the cerebro-spinal fluid. They are to be found in the peripheral blood, but are always scanty, and may appear for two or three days and then be absent for a couple of weeks or more.

The stage lasts for a variable time up to several years, with intermittent pyrexial attacks and increasing anæmia. During its course, intercurrent disease, such as pneumonia, may cause the patient's death. If, however, it does not, the disease terminates in (1) recovery, which is very rare, or (2) the condition known as *sleeping sickness.*

This invariably fatal stage usually lasts from four to eight months before death ends the scene, but may be prolonged up to a year.

There is marked anæmia, with a leucocytosis of large mononuclears.

The pyrexia, previously intermittent and irregular, now becomes of a more constant and hectic type.

Supra-orbital headache is complained of, and is accompanied by pains in the back and chest. The mental condition becomes changed, and symptoms of a chronic meningo-encephalitis are developed.

The face loses its ordinary expression. Laziness, dullness, and apathy increase daily.

Tremors of the tongue and hands are generally present, although paralyses are rare.

There is no anæsthesia, and the superficial reflexes are usually normal. The pupils are equal and dilated.

The increasing asthenia and emaciation are accompanied by progressive drowsiness, until, finally, the patient is almost continually comatose, and incontinence of urine and fæces is established.

At this final stage the temperature becomes subnormal, often to an extreme extent (*vide* chart), and the scene ends with death from secondary bacterial complications, such as purulent meningitis, pneumonia, or generalized suppuration of the lymphatic glands.

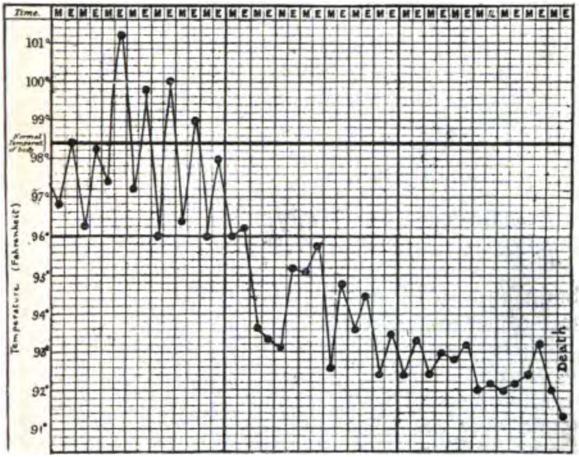

FIG. 28.—HUMAN TRYPANOSOMIASIS. SUBNORMAL TEMPERATURE OF SECOND STAGE.

Treatment. — Serum treatment, chrysoidin, the exhibition of X rays, and other methods, have been tried without success.

So far only two drugs have proved of any value :

1. *Trypan red*, an aniline dye discovered by *Ehrlich* and *Shiga*. It has a marked curative effect on 'caderas'-infected mice. The dose is 5 grains.

2. *Atoxyl*, first used by *Thomas*. It is an organic arsenate, of the formula $C_6H_5NHAsO_2$, and contains nearly 38 per cent. of arsenic in a very non-toxic form.

   This is quite the best treatment so far adopted, and should be given by intramuscular injection of a 5 per cent. solution daily, beginning with a dose of 0·2 gramme of atoxyl, and gradually increasing up to 0·8 gramme. After a fortnight of the larger doses it should be gradually reduced again, and then interrupted for some weeks.

   If given by the mouth, a toxic amount of arsenic may be liberated.

Pathology.—The *lymphatic glands* are enlarged, this being inflammatory and terminating in fibrosis.

There is increased *subarachnoid fluid*, both in the brain and spinal canal, and the lateral ventricles of the former are dilated.

There is injection and inflammation of the *meninges*.

Microscopically, a mononuclear infiltration is found over the whole convex surface of the *brain*, in the sulci, and along the vessels.

The *spinal nerve roots* and the posterior spinal ganglia show inflammatory changes.

The *spleen* is generally enlarged, and often exhibits necrotic areas. There is frequently degeneration of the *bone marrow*.

XL

ULCERATING GRANULOMA OF THE PUDENDA

Definition.—A tropical and infectious granuloma, usually attacking the genitals or contiguous parts.

Synonyms.—Sclerosing pudendal granuloma; perforating granuloma of the thigh.

Geographical Distribution.—The condition was first recognized by *Daniels*, in 1896, amongst natives in British Guiana. Cases are also reported from Southern China, India, North Australia, and Fiji.

Etiology.—The specific virus is unknown. The disease, however, is contagious, auto-inoculable, and venereal in character.

No race and neither sex are exempt, and the condition is usually found between the ages of fifteen and fifty.

Symptoms.—A small and easily-broken nodule begins on or near the genitals, at the side of which a superficial ulcer subsequently forms. This is indolent and very chronic, lasting sometimes for years.

It spreads gradually either by contiguity or continuity, and is attended neither by pain, involvement of the glands, nor constitutional disturbance.

Treatment.—Mercury and potassium iodide are useless. Complete and early excision is the most rational treatment.

Calc. iodide, gr. ii., t. d. s., might be tried internally; and an external application of acid. salicyl. 2 parts, ung. creasoti 30 parts, is sometimes of use.

Pathology.—No caseation occurs, nor are giant cells to be found.

There is a small-celled infiltration of the croium and papillæ.

The epithelium becomes atrophied, the cuticular vessels dilated, and connective tissue takes the place of the cells.

XLI

VITILIGO

Definition.—A disease characterized by the presence of progressive, unraised, white patches.

Synonyms.—Leucodermia ; leucopathia ; piebald skin.

Distribution.—Occurs in various parts of the tropics, and is confined to natives. Probably less rare amongst the Chinese than amongst the Malays.

Indians appear to be the least susceptible.

Etiology.—The disease is unlike albinism, in that it is acquired and not congenital.

It is probably a trophoneurosis.

Symptoms.—Slight pruritus may precede its appearance, which is heralded by an increased deposit of pigment. In the centre of the darkened patch a loss of pigment takes place, and the milk-white area enlarges, driving the pigment before it.

There is no alteration in sensation or secretion, but the hair turns white in the involved areas.

Its progress is a matter of years rather than months.

Treatment.—This is futile.

XLII

YELLOW FEVER

Definition.—An acute specific fever of the tropics, characterized by pyrexia, jaundice and albuminuria.

Synonyms.—Black vomit; yellow jack.

Geographical Distribution.—Primarily a disease of the American continents, it was first definitely described by *Du Tertre* in Guadeloupe, in 1635.

The present endemic centres are—

The littoral of the Gulf of Mexico.
The West Indies.
Brazil.
The West Coast of Africa.

Cases have been imported from time to time into Europe, but have never spread to any extent.

Asia and Australia have been entirely exempt up to the present.

The distribution of the disease is ultimately limited by the distribution of a mosquito—the *Stegomyia fasciata*.

Etiology.—Most of our knowledge of the etiology of yellow fever is due to the work of the French Mission to Brazil in 1903.

Useful work was also done by a U.S.A. Commission in 1900.

The disease is caused by a specific germ as yet undiscovered.

The following have at one time or another been put forward as the cause of the disease:

Cryptococcus xanthogenicus of Freire.
Peronospora luteæ of G. Valle.
Cogumello fungus of Da Lacerda.
Bacillus icteroides of Sanarelli.

These are now only of historic interest.

The germ must be ultra-microscopical, or nearly so, for it will pass through the Chamberland bougie F, but not through the bougie B.

There are certain definite data which should be enumerated:

1. The disease is carried from person to person by the *Stegomyia fasciata*, and, as far as is known, by no other mosquito.
2. After the mosquito's infected meal, twelve days at least must elapse before a subsequent bite can infect a healthy person.
3. Ova laid after the above twelve days can infect the imago adults into which they develop.
4. The serum of a patient is only virulent during the first three days of the disease, and is infective neither subsequently nor during the previous incubation period.
5. Virulent serum will not produce the disease if deposited on a skin abrasion, but must be injected beneath the skin.
6. Virulent serum is quickly rendered innocuous by exposure to the air (for forty-eight hours), or to a temperature of 55° C. (for five minutes).
7. The serum of convalescents possesses both curative and prophylactic properties.

With regard to the *incidence*, every race and age are susceptible, especially in the case of new-comers.

In young children the type is usually mild.

One attack confers immunity.

The disease is in no way contagious.

Symptoms.—The *incubation period* is from three to five days.

The onset is usually sudden, with no prodromata.

Occasionally malaise, headaches, and subjective sensations of mephitic odours, may precede the attack for a few days.

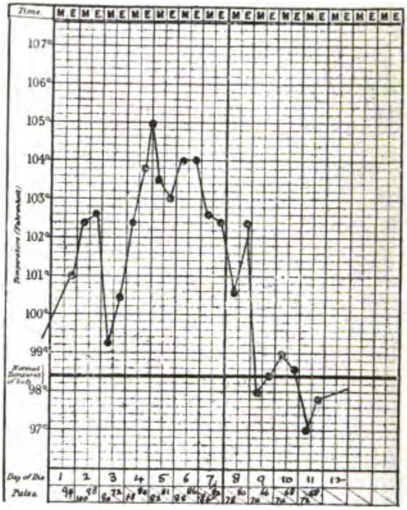

FIG. 29.—YELLOW FEVER: SEVERE CASE; RECOVERY.

A rigor, frequently nocturnal, ushers in a rise of temperature.

There will be a flushed face, injected conjunctivæ, severe frontal headache. Lumbar pains are fairly constant, as also epigastric colicky pain, much aggravated by pressure. Erythema of the genital region is almost a pathognomonic early symptom, and later on may lead to desquamation, ulceration, or even gangrene.

The breathing becomes hurried.

The tongue is furred, with red edges, and later becomes dry and cracked.

There is great thirst, and frequently vomiting of clear and acid fluid. The bowels are constipated.

The urine is much diminished, of acid reaction, and contains plentiful albumen from the second day. The prognosis largely depends on the quantity of the albumen present.

After twenty-four hours' high temperature with full and strong pulse of about 110 or 112, the fever gradually declines until about the third day.

This is the period of remission, and is a crucial point in the disease. There is an abatement of the urgent symptoms, including the gastric irritability. The conjunctivæ, and gradually the whole body, become jaundiced.

In favourable cases the pulse and temperature sink to normal; the volume of the urine increases, and the amount of albumen diminishes.

In unfavourable issues the fever reappears, with much prostration. The pulse throughout is remarkably slow relatively to the degree of pyrexia, resembling in this respect the peculiarity of seven-day fever.

The gastric symptoms become again prominent, and now assume the type of 'black vomit,' which consists of altered red cells and débris, coloured with hæmatin. The albumen in the urine increases, and the amount of urine diminishes.

Pregnant women will always miscarry.

Death, usually from toxæmia or uræmia, occurs between the fourth and tenth day, and is often preceded by a drop in the temperature and a rise in the pulse.

In cases which recover, relapses are rare, but convalescence is protracted, and may be complicated by boils, abscesses, parotitis, or even gangrene.

As a matter of *differential diagnosis*, malaria, blackwater fever, relapsing fever, or infectious jaundice, may have to be excluded.

The ordinary *mortality* varies from 30 to 40 per cent., although it may be much less in a mild epidemic and much greater in a severe one.

Treatment.—There is no specific drug. Nursing is of the utmost importance.

An early purge is useful.

Iced milk and lime-water should be the diet of the first four or five days.

Hyperpyrexia will call for cold sponging.

For the relief of the urgent vomiting, ice should be sucked and a mustard plaster applied to the epigastrium.

Stimulants and digitalis will be required for collapse.

A therapeutic serum, already of acknowledged value, will probably soon be a definite and available realization.

Pathology.—The *blood* is fluid, and the serum coloured with hæmoglobin.

The *liver* shows the most characteristic and important changes. There is no enlargement, but it is soft, yellowish, and anæmic. The intralobular tissues are œdematous, while microscopically there is fatty degeneration and cloudy swelling of the hepatic cells.

The mucous membrane of the *stomach* is softened and congested in patches, the contents usually consisting of the tarry 'black vomit.' The *intestines* contain the same material, and likewise show patchy congestion. The solitary glands and Peyer's patches may be enlarged.

The *kidneys* show a parenchymatous nephritis.

The *spleen* is normal.

APPENDIX I

BIBLIOGRAPHY OF TROPICAL MEDICINE

System of Medicine, vol. ii., part 2 (1907), by Allbutt and Rolleston. (Macmillan and Co. 9×6×2 in. 25s. net.)

The standard large text-book on tropical medicine and parasitology. Illustrated and up-to-date.

Tropical Medicine, Hygiene, and Parasitology (1908), by Brooke. (Griffin and Co. 6½×4×1 in. 12s. 6d. net.)

A concise résumé of the whole subject; of portable size, and illustrated throughout.

Diseases of Warm Countries (1903), by Scheube. (Bale, Sons and Danielsson. 10×6×1½ in. 30s. net.)

From the German. Parasitology scanty. No recent edition. Illustrated.

Tropical Diseases (1907), by Manson. (Cassell and Co. 7½×5×1½ in. 12s. 6d. net.)

Deals with the usual tropical diseases from clinical and practical aspect. Illustrated.

Diseases of Warm Climates (1893), by Davidson. (Pentland. 10×6×2 in. 31s. 6d.)

Is out-of-date. Contains illustrations.

Lectures on Tropical Diseases (1905), by Manson. (Constable. 8½×5½×1 in. 7s. 6d. net.)

The Lane Lectures for 1905.

Fevers in the Tropics (1908), by Leonard Rogers, Professor of Pathology, Calcutta University. (Henry Frowde, Oxford University Press. 10×8×1½ in. 30s. net.)

Contains the Milroy Lectures on Kala-azar, and also much original work on seven-day and other previously unclassified fevers..

Medical Diseases of Egypt, part 1 (1905), by F. M. Sandwith, Consulting Physician to Kasr-el-Ainy Hospital, Cairo. (Henry Kimpton. 8½×5½×1½ in. 7s. 6d.)

Enteric Fever in India and in other Tropical and Sub-Tropical Regions (1906), by E. Roberts. 4to. (Baillière, Tindall and Cox. 21s. net.)

Æstivo-autumnal Malarial Fevers (1901), by C. F. Craig. Demy 8vo. (Baillière, Tindall and Cox. 10s. 6d. net.)

Practical Study of Malaria (Third Edition, 1908), by Stephens and Christophers. (Williams and Norgate. 9×6×1 in. 12s. 6d. net.)

Malarial Fevers (1900), by Thayer. (Henry Kimpton. 8½×6×1 in. 12s. net.)

Treatise on Plague (1905), by Professor Simpson. (Cambridge University Press. 16s. net.)

Leprosy (1895), by Hansen and Looft. (Wright and Co. 8½×5½ in. 10s. 6d.)

Diseases of the Blood (1902), by Coles. (Churchill. 10s. 6d.)

Laboratory Studies in Tropical Medicine (1907), by Daniels. (Bale, Sons and Danielsson. 16s. net.)

Trypanosomes and the Trypanosomiases (1907), by Laveran and Mesnil. (Baillière, Tindall and Cox. 10×6×2 in. 21s. net.)

Animal Parasites of Man (1906), by Braun. (Bale, Sons and Danielsson. 10×7×1 in. 21s. net.)

Parasites of the Domesticated Animals (1906), by Neumann. (Baillière, Tindall and Cox. 9 × 6 × 2 in. 21s. net.)

Monograph of the Culicidæ of the World (1901-6), 4 vols., by Theobold. (Dulau and Co. £4 4s. net.)

Gnats or Mosquitoes (1905), by Giles. (Bale, Sons and Danielsson. 9½ × 6½ × 1 in. 23s. 6d. net.)

The above list does not, of course, represent a tithe of the volumes, monographs, pamphlets, etc., either directly or indirectly bearing on the subject of tropical medicine, but it should be sufficiently comprehensive for practical needs.

Students of tropical medicine can frequently consult with advantage the *Journal of Tropical Medicine*; *Parasitology*, a quarterly journal issued from the Cambridge University Press; the various publications of the Bureau of Government Laboratories, Manila; and also the frequent monographs issued by the Institute for Medical Research at Kuala Lumpur, in the Federated Malay States, and by the Thompson-Yates Laboratory in Liverpool.

APPENDIX II

REGULATIONS OF EXAMINING BOARDS FOR DIPLOMAS IN TROPICAL MEDICINE

(D. T. M.)

UNIVERSITY OF CAMBRIDGE.

The Cambridge Examination takes place twice a year, in January and in August. The following are the requirements:

Any person whose name is on the *Medical Register* is admissible as a candidate to this examination, provided—

I. That a period of not less than twelve months have elapsed between his attainment of a registrable qualification and his admission to the examination.

II. That he produce evidence, satisfactory to the Syndicate, that he has diligently studied pathology (including parasitology and bacteriology) in relation to tropical diseases, clinical medicine and surgery at a hospital for tropical diseases, and hygiene and methods of sanitation applicable to tropical climates.

As evidence of study and attainments a candidate may present to the Syndicate (1) any dissertation, memoir, or other record of work carried out by himself on a subject connected with tropical medicine or hygiene; (2) any certificate or diploma in public health or sanitary science he may have obtained from a recognized examining body. Such evidence will be considered by the Syndicate in determining whether he is qualified for admission to the examination, and by the Examiners in determining whether, if admitted, he shall be included in the list of successful candidates.

The examination will have reference to the nature, incidence, prevention, and treatment of the epidemic and other diseases prevalent in tropical countries. It will comprise the following subjects:

1. The methods of pathological and bacteriological investigation; the examination of the blood; the characters, diagnosis, and life-history of animal and vegetable parasites; the examination, chemical and microscopic, of poisonous or contaminated foods and waters.

2. The origin, pathology, propagation, distribution, prevention, symptoms, diagnosis, and treatment, of the epidemic, endemic, and other diseases of tropical climates, including—malaria; blackwater fever; trypanosomiasis; relapsing fever; dengue; yellow fever; plague; tetanus; beri-beri; dysentery and hepatic abscess, cholera, enteric fever, Malta fever, and specific diarrhœal affections of the tropics; diseases due to cestode and other worms; filariasis; bilharzial disease; specific boils, sores, and other cutaneous affections; mycetoma; ophthalmic affections of the tropics; affections caused by poisonous plants and animals, and by poisonous weapons; sunstroke.

3. The general effects on health in the tropics of seasons and climate, soil, water, and food; personal hygiene, acclimatization; principles of general hygiene, with special reference to food-supplies and water-supplies, sites, dwellings, drainage, and the disposal of refuse; the sanitation of native quarters, camps, plantations, factories, hospitals, asylums, gaols, pilgrim and coolie ships; principles and methods of disinfection.

The examination is partly in writing, partly oral; and partly practical and clinical. The clinical part will be conducted at a hospital for tropical diseases, at which cases will be submitted for diagnosis and comment.

Every candidate is required to pay a fee of six guineas before admission or re-admission to the examination. A candidate who, after being approved for admission, fails to present himself at the examination will not have the fee returned, but will be entitled to present himself without further fee on one subsequent occasion.

Every candidate who passes the examination to the satisfaction of the Examiners will receive from the University a diploma testifying to his knowledge and skill in tropical medicine and hygiene.

UNIVERSITY OF EDINBURGH.

Diploma in Tropical Medicine and Hygiene (*D. T. M. and H.*).—Graduates in medicine and surgery of this University are eligible for examination for the diploma on the expiry of a period of six months after they have obtained these degrees. They must produce evidence of having attended approved courses of instruction in practical bacteriology, including the pathogenic micro-organisms of tropical diseases; in diseases of tropical climates, including the zoological characters and the life-history of disease-carrying insects; in tropical hygiene, and in the clinical study of cases of tropical disease; and certificates of proficiency in the methods of conducting post-mortem examinations, and of preparing reports on them. Regulations for the diploma will be found in the *University Calendar*. The examinations are held in January and July. The fees for the first and any subsequent appearance are: Practical bacteriology, £1 1s.; diseases of tropical climates, £1 1s.; tropical hygiene, £1 1s.; tropical clinical medicine, £1 1s. Total, £4 4s.

UNIVERSITY OF LONDON.

Tropical medicine is now one of the subjects through which graduates of the University of London can proceed to the M.D. Candidates for the degree in this subject must have taken the M.B., B.S. degrees not less than two years previously, unless subsequently to that event they have either conducted a piece of original work approved for the purpose by the University, or have had such special experience as may be similarly approved; in either of these two cases the interval may be diminished to one year. All candidates alike must either have attended for not less than one academic year a complete theoretical and practical course on tropical medicine at a school approved for that purpose, or have been engaged for not less than two years subsequently to taking their degrees in hospital, official, or private practice in regions where tropical diseases prevail. The examination to be passed includes a paper on General Medicine, one on Tropical Medicine, one on Tropical Pathology and

Hygiene, the writing of an essay, and clinical and laboratory tests.

The Schools of Tropical Medicine approved by the University are the London Tropical School of Medicine and the Liverpool Tropical School.

LONDON SCHOOL OF TROPICAL MEDICINE.

The school buildings, laboratories, museum, library, etc., are within the grounds of the Branch Hospital, Royal Victoria and Albert Dock; station, Connaught Road, G.E.R.

There are three courses in the year, each lasting three months, beginning October 1, January 15, and May 1 respectively.

The laboratory, etc., are open daily, and clinical instruction is given daily in the wards of the hospitals. Certificates are granted after examination to those who complete a full course. Resident chambers are available for students, who must be qualified or in their fifth year of medical studies.

An agreement has been concluded between the School and the Royal Veterinary College, Camden Town, by which students of either institution may attend the other; and an interchange of demonstrations has been arranged.

A Craggs Prize of £50 is awarded yearly. All information can be obtained from the Secretary, Seamen's Hospital, Greenwich.

THE UNIVERSITY OF LIVERPOOL.

Diploma in Tropical Medicine (*D. T. M.*).—Ordinance for Diploma in Tropical Medicine:

1. The University shall grant a Diploma in Tropical Medicine (written D. T. M.), which shall be awarded in pursuance of a resolution of the Senate.

2. The diploma shall be awarded only to candidates who possess a qualification to practise medicine recognized for this purpose by the University, and who present satisfactory certificates of having attended approved courses of study, and pass the prescribed examination.

3. Regulations shall determine—

(*a*) The periods and courses of study.
(*b*) The subjects of examination.
(*c*) The fees to be paid for examination.

Regulations for Diploma in Tropical Medicine.—1. The examination for the diploma shall be held three times a year, at the end of the Autumn, Lent, and Summer Terms.

2. Candidates, before presenting themselves for the examination, and subsequent to having obtained the recognized qualification to practise medicine, shall have attended—

(*a*) A three months' course of study in tropical pathology and hygiene in the University.
(*b*) A course of instruction in a hospital recognized by the University, in which beds are specially reserved for tropical diseases.

3. The subjects of examination shall be :

(*a*) Tropical pathology and parasitology.
(*b*) Tropical and applied bacteriology.
(*c*) Tropical hygiene and sanitation.
(*d*) Tropical medicine, including etiology, symptoms, diagnosis, and treatment of tropical diseases.

4. The fee for the examination for the diploma shall be £5 5s.

(For outline of courses, see prospectus of the Liverpool School of Tropical Medicine.)

As much practical work as possible should be done by the student, both in the laboratory and in the wards of a hospital.

As a useful auxiliary to home reading, Messrs. Charles Baker, of 244, High Holborn, have, at my suggestion, listed two sets of microscopical specimens, covering most of the ground of tropical medicine.

Baillière, Tindall & Cox, 8, *Henrietta Street, Covent Garden.*